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DISEASES OF THE HEART

SIR JAMES MACKENZIE

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OXFORD MEDICAL PUBLICATIONS

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BY

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PREFACE TO THE FOURTH EDITION

This book is written in the profound conviction that the conception of research, which is dominant to-day, is inadequate for the solution of many medical problems. The reasons on which this conviction is based appear in the body of this book.

Here in this Preface little need be said, but a few general observations may not be out of place.

It behoves every investigator to pause from time to time to see if the line he is pursuing is leading towards the attainment of the object for which he set out. Otherwise it may be found that, though he may be bringing to light many new and important facts, he is no nearer attaining the object of his research. He may even be following a path which does not lead to the attainment of his object.

When I wrote the last edition of this book, I had become so deeply interested in the line of research I had pursued for years, that I fancied I had been making a considerable advance. For several years I could not see my way to improve upon the last edition, and therefore, when it was exhausted, I did not attempt to rewrite it, but was content to issue from time to time a series of reprints with minor revisions.

But, of late years, doubts began to arise in my mind as to the sufficiency of the researches that had been made, and which were still being made by myself and others. On clearly defining the object of all these researches, it became evident that, notwithstanding the many new phenomena that were being brought to light by the devising of ingenious methods, no progress was being made towards attaining the great object of all our undertakings. This edition is entirely rewritten.

The main duty of a doctor engaged in treating the sick is to combat ill-health and death. To do this with a prospect of success it is necessary

to know the nature of ill-health and the conditions which lead to death. It is manifest that without this knowledge the doctor's attempts to combat ill-health and death would be but shooting in the dark. It is incumbent upon those of us who seek to instruct the doctor in the performance of his duty to provide him with the kind of knowledge which will enable him clearly to understand the nature of the conditions he is expected to combat. When, then, we put to ourselves the question, what is the nature of the phenomena of ill-health, we must confess that our ideas are far too vague to give a definite answer to the question.

In the sick man we may detect a great many phenomena which are indications of ill-health, but it must be admitted that we do not know how these phenomena are brought about. This becomes apparent when we analyse our knowledge of the simplest sign or symptom of disease.

Ill-health is recognized by the presence of certain sensations felt by the patient, or by signs which the doctor detects—these are the symptoms of disease.

A symptom is a manifestation of unusual or abnormal activity of an organ. In the production of this manifestation there are other structures involved than those which exhibit the manifestation. It is manifestly necessary for our purpose that we should know what are those other structures, and particularly in which of those structures the disturbance arises which results in a given symptom. Those structures are composed of living cells, and the cells have particular functions to perform in the maintenance of health. In ill-health these functions are modified. Therefore, for the due understanding of the nature of a symptom, it is necessary, not only to recognize the structures which take part in its production, but also the nature of the modified functions which lead to the symptom.

It is thus seen that there are various factors concerned in the production of a symptom; and the state of knowledge which has resulted from all the investigations that have taken place has failed to reveal the factors concerned in the production of any symptom, however simple and obvious it may be.

This defect in knowledge is not peculiar to cardiology.

In the endeavour to obtain a better understanding of ill-health, clinical medicine has been broken up into a great many branches. The purpose of this division was supposed to be the more thorough investigation into particular kinds of disease. As a consequence there have been devised innumerable methods for the detection of new symptoms, and this procedure has resulted in the accumulation of an enormous mass of symptoms. In spite of this, clinical medicine has failed to provide the knowledge necessary for intelligent practice.

This has been dimly recognized, and in recent years attempts have been made to remedy this defect in knowledge by employing what are considered more scientific methods, by adopting the methods of the experimental branches of medicine. But it has not been recognized that the defect which is fatal to progress in clinical medicine is also fatal to progress in experimental medicine.

The methods employed by the bacteriologist, the pharmacologist, and the physiologist all result in the production of reactions. The great bulk of the mechanical devices have been devoted to the recording of these reactions with great precision, and because of this the impression has been created that scientific accuracy was being attained. This belief has led to the adoption of many of these devices in clinical medicine.

But it will be observed that the experimental investigators do not get beyond the recognition of a reaction. A reaction is exactly of the same nature as a symptom. There are factors concerned in the production of every reaction which are identical with those which produce a symptom. When the matter is looked into it is found that the experimentalist stops short at exactly the same place as the clinician. The next step to be pursued is common to both.

This is brought out if we take a simple instance common to all branches of medicine, clinical and experimental, such as an increase in the pulse-rate. To-day no one can tell what are the factors concerned in producing this manifestation.

The custom has been handed down from past ages that on the discovery of a new phenomenon a name must be found for it. The most common method which has guided the finding of a name was to use one

which was based on the superficial resemblance of the phenomenon to something that was present in the investigator's mind.

The methods of investigation pursued hitherto have led to the accumulation of a mass of symptoms and reactions. Most investigators end by adding to this mass and by introducing some new term. So confused is this mass that it is beyond the comprehension of any individual, and one result is that it effectually obscures the path of progress, so that the investigator himself raises a barrier to further progress.

As all investigators are practically dealing with the same phenomena, and as the investigator in each branch sees the phenomena under different circumstances, each one applies a name which meets his own notion. The result is that the worker in one field is unable to understand the language of workers in other fields, although they are all dealing with the same kind of phenomena. There is thus lost that community of ideas and co-ordinate participation in work which is so essential to progress. The question arises—how can medical investigation be carried beyond this stage? Manifestly by understanding the factors concerned in the production of the symptom or reaction. The need for doing so has indeed been recognized, but unfortunately it took the form of vague speculation.

Reference has just been made to the increase in pulse-rate. To indicate this increase the term ' stimulation ' is employed, but when we inquire as to what is meant by stimulation, the information provided simply refers to the evidence of increased activity. What structures were stimulated, and as to what was the nature of the process called ' stimulation ', we get no information. It is the same in regard to other terms in common use which indicate any manifestation of activity, such as inhibition, conduction, contraction, tone, and so forth.

This latter conception is the one which has emerged from the investigations carried on at the St. Andrews Institute for Clinical Research.

Before entering upon our work we reviewed the attitude of research towards the diseases common among the people, and we found that the conceptions of research hitherto employed were inadequate for the solution of problems met in general practice. We found that this was due to the stopping of all forms of research at the discovery of the symptom. We then sought for some principle that would lead us to a better under-

standing of symptoms, and there emerged one which guided us to the recognition of the factors concerned in the production of the symptom.

It has hitherto been considered that to go beyond the stage of recognizing a symptom or a reaction was to probe into the origin of life and to speculate about colloids and electrons. But this is not so: there is a vast intermediate field to be explored.

The factors concerned in the production of a symptom are structures definitely constituted of living cells which are capable of recognition. These cells perform definite functions, and there is a great deal to be learnt about the manner in which they perform these functions individually and when collected together to form an organ.

The inquiry into these matters has opened up a new field, and has thrown an unexpected light upon the nature of functional activity.

In this book there are set forth the principles which govern this line of research, and the results are given of its application to the interpretation of cardiac phenomena, and it will be found to carry us a long way forward.

It is not contended that the principles of research described here are beyond criticism, or that they are final. They only represent the state of our knowledge at the present time. A fuller knowledge may require their modification or supersession. In any case, they are an improvement upon the present conceptions.

J. M.

January 24, 1925.

My husband had finished the above draft of the Preface shortly before he died. He had already read the proofs of this new edition, and Dr. James Orr, of St. Andrews, one of my husband's colleagues on the staff of the St. Andrews Institute of Clinical Research, has been good enough to go over the proofs again and to verify the references and tracings. I have to thank him for his great kindness and for the attention he has given to the final preparation of this book for the press.

My thanks are due also to Dr. Laing, who has prepared the index.

FRANCES B. MACKENZIE.

London, *July* 1925.

PREFACE TO THE FIRST EDITION

In the following pages are given the results of observations on affections of the heart, made during an active practice of over a quarter of a century. As the nature of the heart affection can only be inferred from the presence of one or more symptoms, my special object has been to ascertain the mechanism by which the symptoms are produced, to find out their relationship to organic changes in the heart, to ascertain their prognostic significance, and, finally, to employ them as a guide for treatment.

This line of observation has revealed many new and unexpected features, and has necessitated the employment of special methods and the watching of individual cases for many years.

To make the results of use in practice it has been found necessary to enter into considerable detail, and it has been difficult to make the account at the same time succinct and intelligible. A certain amount of detail is necessary, yet too much would be wearisome and perhaps confusing, hence controversy has been avoided and the observations have been given with the explanation that seems most reasonable.

Many methods of examination that occupy prominent places in text-books have been briefly dealt with or even ignored, not because I do not recognize their usefulness in certain cases, but because my object has been to deal with matters of practical value in the every-day examination of patients. It may appear that an unnecessary amount of attention has been devoted to details, many of which can only be recognized by special apparatus. But to bring conviction proof had to be elaborated. Many of the seemingly trivial signs, such as the minute differences in the size of a pulse-beat, or a slight delay between the auricular and ventricular systoles, are really of vital importance in revealing changes which have been proved to be due to very definite affections of the heart. In the

same manner the study of irregularities is of the greatest service, as their presence is easily detected and their significance has never been properly understood. A close study of irregularities throws an unexpected light upon the functional derangements of the heart, and affords grounds for an intelligent diagnosis upon which a rational treatment and prognosis can be based.

The main purpose of all my work has been to obtain a guide for treatment, and my readers may be disappointed at the seeming barrenness of my work in this respect. A careful search has been made for the essential principles that should govern a rational treatment, and if only few drugs and suggestions are given, it is because the treatment of cardiac disease at present in vogue requires careful revision in the light of the more accurate diagnosis now made possible by means of the graphic method of examination.

In routine practice it is not usually necessary to take graphic records. If one is trained to make careful and minute observations by the ordinary methods, and to have these checked by graphic records, one can ultimately acquire the power of recognizing the majority of movements of the circulation without graphic records. In regard to the tracings given in this book, a selection has been made from an enormous number of observations, and are rarely exceptional, but are types of the commoner forms. The interpretation of these records may prove to be faulty, and an endeavour has therefore been made to keep the actual observation separate from the interpretation, so that if the latter be erroneous, the recorded movements may at least remain available for future workers in this field.

It was originally intended to give a more complete account of the morbid anatomy of the heart, and for that purpose Professor Keith has investigated a great number of hearts, of which I have kept careful clinical records, but the investigation, so far, has brought to both of us the conviction that before the pathology of the heart can be put on a satisfactory basis a more thorough and minute examination of the post-mortem appearances in correlation with the clinical symptoms is necessary. The post-mortem aspect of heart disease is therefore dealt with very briefly, and my observations in this respect are intended to be suggestive rather than conclusive.

I had intended to give the means of judging the state of the heart

in cases apart from those directly due to affections of the heart, as in fevers, pregnancy, surgical diseases, diseases of other organs, the administration of chloroform. This has only been carried out to a limited extent, because, when the results came to be analysed, I did not see my way clear in all cases to give satisfactory guides, so that many observations have been omitted. I hesitated to include my observations on chloroform administration, because the matter is still very vague. I feel convinced that the reason chloroform is attended with so much danger will never be solved until prolonged and painstaking investigation is made during its administration along the lines of observation detailed in this book, and I leave my remarks in this unsatisfactory state in the hope that others may solve the question.

What a tool is to a workman, so should a text-book be to the busy practitioner. In cases of heart affection one or two symptoms are usually most prominent, and by giving clear definitions of the terms employed, and by arranging the index and the discussion in the text, an endeavour has been made to facilitate the rapid inquiry into the meaning of any given symptom.

J. M.

London, W.,
September 1908.

CONTENTS

INTRODUCTION

PAGE

The science of medicine. The need for a new outlook in medicine. The assessment of the value of symptoms. The simplification of medicine 1

CHAPTER I

A Review of Modern Cardiology 5

CHAPTER II

The Significance of Symptoms

Diagnosis. The object of a prognosis. Prognosis and treatment. Modern medicine and prognosis. Prognosis in the past. The effects of unproved beliefs 12

CHAPTER III

The Principles of Prognosis

The neglect of prognosis. The importance of the trained senses. Research into prognosis. A new method for investigating prognosis. Sources of prognostic symptoms in heart affections 17

CHAPTER IV

The Principles of Prognosis (*continued*)

The reflex arc and prognosis. Prognosis and the multitude of symptoms. The kind of prognosis wanted. Prognosis and heart failure. The manner of death 23

CHAPTER V

On measuring the Functional Efficiency of the Heart . 27

CHAPTER VI

Heart Failure and the Response to Effort

The rest and reserve power of the heart. The laws of 'all or nothing' and fluctuation. The response of the heart to effort. The origin of symptoms of heart failure. Symptoms of heart failure due to a deficient output of blood: (*a*) Breathlessness; (*b*) Orthopnœa; (*c*) Cheyne-Stokes breathing; (*d*) Cardiac asthma; (*e*) Effects of diminished blood supply to the brain; (*f*) Dropsy; (*g*) Œdema of the lungs; (*h*) Enlargement of the liver; (*i*) Cyanosis 30

CHAPTER VII

The Principles of Investigation

PAGE

The problem of ill-health. The end result. The need for a new concept. The factors concerned in vital activities 36

CHAPTER VIII

The Reflex Arc

The constitution of the reflex arc. The meaning of the reflex arc. The application of the principle of the reflex arc. The muscular and genetic systems of the heart. The part played by the genetic system. The application of the reflex arc. The application of the reflex arc to the interpretation of heart irregularities. The value of the interpretation of symptoms by means of the reflex arc 38

CHAPTER IX

The Vital Processes

Introduction. Definition of the term ' vital process '. The cell impulse and its investigation. The basis for a classification of symptoms 48

CHAPTER X

The Vital Activities in Plants

The method of investigating vital activities. The principles on which the investigation is based. The functions of plant cells: Stimulation; Contraction; Conduction; Inhibition; Control. The cell impulse 51

CHAPTER XI

The Vital Activities in the Human Body

The contraction of muscle. The structure and function of nerve cells. The genetic system of the heart. The functions of the sympathetic and vagus nerves. The function of motor nerves. The impulse, the nerve, and the reaction. The influence of chemical agents. Control. Controlled and uncontrolled movements in the medusa . . . 55

CHAPTER XII

The Vital Activities in the Human Body (*continued*)

Fluctuation. Tone. Centres. The action of drugs and other chemical agents. The manner of death. The cell impulse and electricity 64

CHAPTER XIII

The Classification of Symptoms

The object of a classification. The basis of a classification. The principles of classification of the symptoms of disease. Classification and investigation. The application of classification to investigation 73

CHAPTER XIV

The Role of the Capillaries in the Production of Symptoms

PAGE

The capillary as an organ. The regulation of capillary activity. Nerves and the capillaries. Capillaries controlled. Capillaries uncontrolled and failure of the circulation. The capacity of the capillary reservoir. The cause of the failure of the circulation due to the capillaries. The obliteration of capillaries. Effects produced by obliteration of the capillaries. The cause of capillary obliteration. The obliteration of the capillaries in the eye 77

CHAPTER XV

The Heart-beat

The parts concerned in the heart-beat: (*a*) The genetic system; (*b*) The muscular system. Development of the heart. The relation of the genetic to the muscular system . . 84

CHAPTER XVI

Anatomy of the Genetic and Muscular Systems

The structure of the sino-auricular node. The structure of the auriculo-ventricular node. The structure of the auriculo-ventricular bundle. The muscle of the heart. The blood supply of the heart. The contraction of the cardiac chambers 90

CHAPTER XVII

The Nerve Supply of the Heart

The afferent system of nerves. The sympathetic and vagus nerves. The relation of the sympathetic and the vagus nerve to the heart. Reciprocal innervation. The denervated heart. Stimulation of the vagus. Stimulation of the sympathetic 93

CHAPTER XVIII

The Function of the Genetic System of the Heart

Introduction. The generation of impulses. The rhythmogenic function. Conductivity. Control. The part played by the nerves in control. The impulses which stimulate the contraction of the heart. The law of 'all or nothing' 98

CHAPTER XIX

Classification of the Variations in Rate and Rhythm of the Heart

The advantages of a classification. Variations due to increased excitability of different parts of the genetic system. Variations due to decreased excitability of different parts of the genetic system. Variations due to the cessation of activity of parts of the genetic system. Table and diagram of the abnormal rhythms 104

CHAPTER XX

The Place of Instrumental Methods

The use of graphic and electro-cardiographic records. The training of the senses . . 108

CHAPTER XXI

The Normal Rhythm

PAGE

The liability of the s.-a. node to disturbance. The normal rate of the heart. The increased pulse-rate. The significance of the increase in the pulse-rate. The rate of the heart as a test of cardiac efficiency. Diminished frequency with the normal rhythm. Differentiation. Differentiation by the unaided senses. Prognosis 110

CHAPTER XXII

Disturbances of the S.-A. Node

The youthful type of irregularity. Periodic variation in rhythm. Standstill of the whole heart. Other sinus irregularities. Palpitation. Prognosis of the disturbances of the s.-a. node 117

CHAPTER XXIII

The Independence of the Different Parts of the Genetic System 125

CHAPTER XXIV

Variations due to increased Excitability of the Genetic System

The extra-systole. Clinical methods of investigating the extra-systole. Experimental methods of investigating the extra-systole. The genetic system and the extra-systole. Different forms of extra-systole. The electro-cardiogram of the extra-systole. The irregularity produced by the extra-systole 128

CHAPTER XXV

Variations due to increased Excitability of the Genetic System (*continued*)

The size of the extra-systolic pulse-wave. Comment upon the origin of the extra-systole. Clinical diagnosis. Prognosis. Treatment 139

CHAPTER XXVI

Variations due to increased Excitability of the Genetic System (*continued*)

Paroxysmal Tachycardia

The nature of the attacks. The different forms of paroxysmal tachycardia. The small beats. Clinical diagnosis. Prognosis. Treatment. Observation 2 147

CHAPTER XXVII

Auricular Fibrillation
(Synonyms : Paralysis of the Auricle, Nodal Rhythm)

Historical account. The term auricular fibrillation 158

CHAPTER XXVIII

The Diagnosis of Auricular Fibrillation

PAGE

Appearance of the patient. The distress in breathing. The pulse. The sounds. The jugular pulse. Graphic records. The radial pulse. The jugular pulse. Fibrillation waves. The electro-cardiogram 162

CHAPTER XXIX

Cases illustrative of Auricular Fibrillation

Observations 5-17 170

CHAPTER XXX

The Origin and Nature of Auricular Fibrillation

Distinctive features of auricular fibrillation. The disorderly activity of organs. Disease of the s.-a. node. The age incidence of auricular fibrillation. The relation of heart-block to auricular fibrillation. A comment on the origin of auricular fibrillation . . . 181

CHAPTER XXXI

The uncontrolled Auriculo-Ventricular Node

The independent a.-v. node. The effects of loss of control. The relation of the a.-v. node to the fibrillating auricle. The a.-v. node in the tortoise heart. Stimulation and inhibition of the a.-v. node. The value of the sphygmogram. The pulse irregularities of auricular fibrillation. The a.-v. node and the contraction of the ventricle. The nature and origin of the small beats. The disappearance of the small beats. The relation of heart failure to the small beats. The imperfect control of the independent a.-v. node 187

CHAPTER XXXII

The Production of Heart Failure in Auricular Fibrillation

Heart failure in auricular fibrillation. The mechanism of heart failure. The symptoms of heart failure. Sudden recovery from heart failure. Angina pectoris and auricular fibrillation 196

CHAPTER XXXIII

Prognosis of Auricular Fibrillation

The principles of prognosis in auricular fibrillation. Variable results of auricular fibrillation. The condition of the muscle. Valve disease. Response to treatment. The degrees of heart failure. Sudden death 199

CHAPTER XXXIV

Treatment of Auricular Fibrillation

General. The use of digitalis. Method of administration. Standardization of drugs. The employment of digitalis. Sudden death from digitalis. Treatment by quinidine 204

CHAPTER XXXV

Auricular Flutter

PAGE

The recognition of auricular flutter. The diagnosis of auricular flutter. The patient's complaint. The inspection of the jugular pulse in auricular flutter. The diagnostic character of the radial pulse in auricular flutter. The pulsus alternans and irregular pulse in auricular flutter. The jugular pulse. The electro-cardiogram 215

CHAPTER XXXVI

Cases illustrating Auricular Flutter

Observations 22-36 221

CHAPTER XXXVII

The Origin and Nature of Auricular Flutter

The origin and nature of auricular flutter. The 'circus rhythm'. The nature of the contraction in auricular flutter. The impulses discharged by the auricle. Origin of the paroxysmal attacks 234

CHAPTER XXXVIII

Heart Failure in Auricular Flutter and Prognosis

Syncopal attacks. Respiratory symptoms. Prognosis 238

CHAPTER XXXIX

Treatment of Auricular Flutter

Digitalis. Danger of digitalis. The fibrillation that follows auricular flutter. Observations 241

CHAPTER XL

Decreased Excitability of the Genetic System (Heart-Block)

Introduction. Definition of heart-block. The inter-systolic period. The vital process concerned in delaying the passage of impulses. The *a-c* interval. Partial heart-block. Complete heart-block. The effect of the ventricular systole on the auricular. Conditions inducing heart-block. The effect of effort. The effect of atropine. The size of the ventricular contraction in heart-block. Sino-auricular block. Bundle block. Arborization block. Heart-block associated with other diseases. The age incidence . 249

CHAPTER XLI

Decreased Excitability of the Genetic System (*continued*)

Clinical diagnosis of heart-block. Heart failure and the symptoms produced by heart-block. Prognosis. Treatment 261

CHAPTER XLII

Cases illustrative of Heart-block

Observations 39-61 265

CHAPTER XLIII

The Pulsus Alternans

PAGE

Conditions favouring the appearance of the pulsus alternans. Clinical diagnosis. Differential diagnosis. Prognosis 284

CHAPTER XLIV

Combined Affections of the Genetic System

Auricular fibrillation and heart-block. The effect of drugs. The vagus effect upon the genetic system 290

CHAPTER XLV

Cases illustrative of the Combined Affections of the Genetic System

Observations 63-68 297

CHAPTER XLVI

The Genetic System in the Production of Death

The suspension of the activity of the conducting system. Ventricular fibrillation . . 304

CHAPTER XLVII

Affections of the Myocardium

Nature of the symptoms. Increased size of the heart. Dilatation of the heart. Effort and dilatation. Dilatation and heart failure. Muscle failure. Prognosis. Treatment . 306

CHAPTER XLVIII

Cases illustrative of Affections of the Myocardium

Observations 69-73 314

CHAPTER XLIX

Syphilitic Affections of the Heart 319

CHAPTER L

Angina Pectoris

Introduction. Vital processes. Stimulation by mechanical means. Stimulation by vital processes. Impulses from muscles. Pain from muscle. Stimulation. Hyperalgesia. Experimental hyperalgesia. Increased excitability of the nervous system. Two forms of angina pectoris. The localization of pain. The value of pain in diagnosis. The value of pain in prognosis. Angina pectoris and the principle of the reflex arc . . 324

CHAPTER LI

Primary Angina Pectoris

The oncoming of angina pectoris. Attacks of angina pectoris. The compression of the chest. The sense of depression. Vaso-motor disturbances. Increased flow of saliva. Vomiting. Gaseous eructation. Increased flow of urine. Manner of death. . . 334

CHAPTER LII

Angina Pectoris (*continued*)

PAGE

Prognosis. Treatment. Treatment for attacks of pain. Treatment for the prevention of attacks of pain. The surgical treatment of angina pectoris 340

CHAPTER LIII

Angina Pectoris (*continued*)

Secondary angina. The significance of the secondary form of angina. Prognosis. Treatment 348

CHAPTER LIV

The Heart in Debility and the Soldier's Heart

'Debility.' Differential diagnosis 351

CHAPTER LV

Sounds and Murmurs, Normal and Abnormal

The part played by the valves in producing ill-health. The significance of murmurs. The attitude of the profession to abnormal sounds. Variations in the sounds of the heart. The cause of functional murmurs. The quantity of blood thrown back to cause a murmur. The differentiation of functional and organic murmurs. The significance of functional murmurs. The significance of organic murmurs 354

CHAPTER LVI

Valvular Defects

The manner of heart failure with valvular defects. Mitral stenosis: Conditions inducing heart failure in mitral stenosis; The murmurs present in mitral stenosis; The mid-diastolic murmur; Progress and symptoms of mitral stenosis; Occasional symptoms. Mitral regurgitation: Murmurs due to mitral regurgitation; Conditions inducing heart failure in mitral regurgitation 362

CHAPTER LVII

Valvular Defects (*continued*)

Affections of the tricuspid valves: Tricuspid incompetence; Tricuspid stenosis. Disease of the aortic valves: Ætiology; Aortic stenosis; Aortic regurgitation. Prognosis in valvular affections. Treatment 370

CHAPTER LVIII

Adhesive Mediastino-Pericarditis

Ætiology. Symptoms. Prognosis. Treatment 377

CHAPTER LIX

Congenital Affections of the Heart

Ætiology. Symptoms. Prognosis. Treatment 380

CHAPTER LX

Aneurysm and Dilatation of the Aorta

PAGE

Aneurysm: Introduction; Definition; Cause; Arteries affected; The different kinds of aneurysms; Symptoms of aneurysm of the thoracic aorta; Aneurysm of the ascending aorta; Aneurysm of the transverse aorta; Aneurysm of the descending aorta; Aneurysm of the abdominal aorta; Prognosis; Treatment. Dilatation: Symptoms of dilatation of the aorta; Prognosis; Treatment 382

CHAPTER LXI

The Heart in Acute Affections

Introduction. Manner in which the heart is affected in infections. Symptoms in myocarditis. Symptoms in endocarditis. Symptoms in pericarditis. The heart in rheumatic fever. The heart in pneumonia. The heart in diphtheria. Septic infections. Prognosis. Treatment 390

CHAPTER LXII

The Principles of Pathology and of Treatment . . 401

CHAPTER LXIII

The Examination of the Patient

Methods of examination. The sense of exhaustion and fainting: Exhaustion in ill-health; Differentiation; The mechanism of exhaustion; The clinical significance of the sense of exhaustion; Exhaustion due to an ineffective heart-beat; Exhaustion due to vaso-motor disturbances; Exhaustion and the response to effort; Fainting . . 409

CHAPTER LXIV

Instrumental Methods of Examination

The sphygmograph. The polygraph. The clinical polygraph. The ink polygraph. The electro-cardiograph. The electro-cardiogram 418

CHAPTER LXV

The Position and Movements of the Heart

The position of the heart. The standards for recognizing the events in a cardiac revolution. Conditions of the chest-wall permitting the recognition of certain movements of the heart. The nature of the movements graphically recorded. The apex beat. Interpretation of a tracing of an apex beat due to the systole of the left ventricle. The auricular wave. Retraction of yielding structures in the neighbourhood of the heart during ventricular systole. Liver movement due to cardiac aspiration. Epigastric pulsation. The apex beat due to the right ventricle. Significance of the inverted cardiogram. Alteration of the apex beat due to retraction of the lung. The shock due to the ventricular systole 429

CHAPTER LXVI

Examination of the Arterial Pulse

PAGE

Superiority of the digital examination. What is the pulse? Inspection of the arteries. Digital examination of the arteries: The condition of the walls; The size of the artery; Character of the pulse; The pulse-rate; The size of the pulse-wave; The impact of the pulse-wave on the finger; The rhythm of the pulse; The two radial pulses compared. The value of a sphygmogram. Definition of a sphygmogram. Events occurring during a cardiac revolution revealed by the sphygmogram. Features of the sphygmogram due to instrumental defect 445

CHAPTER LXVII

Arterial Pressure

The cause of arterial pressure. Methods of measuring the blood-pressure. Increased blood-pressure. Hyperpiesis. Effect on the heart of increased peripheral resistance. Increased arterial pressure and heart failure. Prognosis of high blood-pressure. Treatment of high arterial pressure. 453

CHAPTER LXVIII

The Venous Pulse

What the venous pulse shows. Inspection of the jugular pulse. Methods of recording the jugular pulse. The recognition of the events in a jugular pulse. Description of the events in a cardiac cycle. The causes of the variation of pressure in the auricle and in the jugular vein. Standards for interpreting a jugular pulse. The carotid wave. The notch on the ventricular wave. The diastolic wave. Changes due to variation in the rate of the heart. Method of analysing a tracing. The ventricular form of the venous pulse. Anomalous forms of the venous pulse. Conditions giving rise to a venous pulse 461

CHAPTER LXIX

Enlargement and Pulsation of the Liver

The relation of enlarged liver to heart failure. Reflex symptoms. Signs of enlargement of the liver. Pulsation of the liver. Conditions producing enlargement and pulsation of the liver. Jaundice. Different forms of the liver movement. Differential diagnosis of liver enlargement. Prognosis. Treatment 477

Index of Names 485

General Index 486

DISEASES OF THE HEART

INTRODUCTION

The science of medicine.
The need for a new outlook in medicine.
The assessment of the value of symptoms.
The simplification of medicine.

It may help the reader to understand the main motives which influenced the presentation of the matter in this book to make brief reference to the objects aimed at apart from the specific subject of diseases of the heart. These are (1) to recognize the position of medicine among the sciences ; (2) to show the need for a new outlook in medicine ; (3) to indicate the highest object of medical investigation, namely, the assessment of symptoms ; and (4) to simplify medicine by rendering the phenomena of ill-health easy of comprehension by the rigid adherence to fundamental principles.

It is not presumed that these objects have been fully attained. What has been done consists mainly in calling attention to their importance and in indicating some steps that have been taken to achieve these objects. It is necessary to recognize that if ever the practice of medicine is to be rendered intelligent it must be along some such lines that progress can be made.

The science of medicine.—To a certain extent the confusion of mind which hampers progress in medicine has come about through a failure to appreciate the position of medicine among the subjects dealing with natural phenomena. It has not been recognized that medicine is a subject with aims of its own and methods of its own as distinct from other subjects as botany is from chemistry or astronomy. The idea has been fostered that in order to render medicine 'scientific' the methods and devices of other sciences must be employed. One might as well say that to render botany scientific the methods of the chemist must be employed. No doubt the chemist can contribute something of value to the science of botany, but botany must nevertheless pursue its own methods, though using the methods of other sciences to help to solve some of its problems.

It is the same with medicine—the methods of other sciences may be employed to help in the solution of its problems, but if medicine is to make progress it must use its own methods, which are independent of those of other sciences.

A fallacy prevalent to-day is that certain departments belong to the 'scientific side of medicine'. Because bio-chemistry or physiology or bacteriology may be considered sciences as branches of biology, the part they play in medical affairs does not render these subjects 'scientific' branches of medicine. At the best these subjects may contribute some portion of knowledge which the medical investigator may use, but the real problems of medicine are beyond their scope, and they can only play the part of providing some of the knowledge which medicine uses—to medicine they can only be hewers of wood and drawers of water.

It is the failure to recognize this which has led medicine astray, and progress will be gravely hampered until it is recognized that for the solution of its problems medicine must recognize that its objects and methods are peculiar as well as its opportunities.

To obtain a knowledge of disease, the circumstances which favour or induce its onset, the earliest symptoms of its presence, the modification of these symptoms as it progresses, with all the variations which occur till its termination in death or recovery, must be studied. It is manifestly impossible to combat disease effectively without this kind of knowledge, and it can be obtained only by those who have the opportunity of watching individual patients through the course of an illness. It is manifest that this is beyond the range of the so-called 'scientific departments'. In this book an endeavour is made to indicate the opportunities, and the principles that should be employed, to raise medicine to the dignity of a science.

The need for a new outlook in medicine.—In this book an endeavour is made to present the subject of affections of the heart in such a manner that certain fundamental principles will be exhibited which are essential, not only to the understanding of the phenomena of ill-health produced by the heart, but by every organ of the body. The need for such a presentation will become apparent to any one who scrutinizes the progress of medicine in recent years. If such a survey be undertaken the remarkable fact emerges that, notwithstanding the widespread interest taken in medical research and the enormous amount of labour spent upon it, the most obvious of all problems, whose solution is necessary for the rational practice of medicine, remains practically untouched.

Every patient who consults a doctor does so because he feels ill, yet if we ask what is ill-health? or what is the nature of its phenomena? we shall ask in vain, for nowhere is there to be found an understanding of the nature of the simplest phenomenon of this kind.

The hampering effect of the absence of this kind of knowledge upon medical progress is seen in every field. Crusades and campaigns are being organized against diseases whose effect upon the body is not understood, beyond the fact that they produce certain symptoms, and that death eventually results.

Manifestly, if we are to combat a danger we must know the nature of the danger, and while we know that tuberculosis, cancer, and heart disease produce ill-health and death, we do not know *how* they do it. When an enterprise is

undertaken which requires a particular kind of knowledge, and it is assumed that this knowledge exists when, in fact, it does not exist, it does not require the gift of prophecy to foretell the end of such an enterprise, and thus it is that the path of medicine is strewn with enterprises that have failed. Notwithstanding the great number of people engaged in research and the energy with which it is pursued, the phenomena of disease are still so little known that the need for a better concept to guide research becomes urgent.

The purpose of the doctor in the examination of his patient is to detect the signs and symptoms which reveal the nature of the patient's infirmity. These afford the doctor a knowledge by which he should be able to recognize what steps to take to remove the cause, to stay the progress, or to mitigate the ravages of the disease from which the patient suffers.

The phenomena of ill-health are what are recognized as the signs and symptoms of disease. Disease can only be recognized by the signs and symptoms which it produces, and the diligent examination of patients enables a great many of these to be recognized. Hitherto a great deal of research in clinical medicine has been spent in devising methods for detecting the signs and symptoms of disease.

The assessment of the value of symptoms.—Much more is required of the doctor than the detection of signs and symptoms. To assess their value is by far the most important of the doctor's duties. It is comparatively easy to detect a sign, but it is a matter of infinite difficulty to assess its value, and it is here where medicine breaks down.

To assess the value of a symptom is to understand its relation to those vital processes upon which the life and health of the individual depend. This requires a knowledge of the processes and structures whose impairment induces ill-health and imperils life. Without this knowledge the assessment of the value of the symptom can only be a matter of conjecture or guessing. Such guessing is far more likely to be wrong than right, and as a consequence the practice of medicine is rendered not only inefficient, but also often results in the complete misunderstanding of the patient's complaint.

The simplification of medicine.—For the intelligent practice of medicine and the understanding of disease the simplification of medicine is necessary. So long as medicine requires an increasing number of specialists to interpret the phenomena of disease it may be taken for granted that the subject, though becoming ever more and more complex, is not necessarily making progress.

I hold the view that the phenomena which at present are so difficult of comprehension on account of their number and diversity, are all produced in a few simple ways, and that with their recognition what is now so complex and difficult will become simplified and easy to understand. This means a recognition of principles and a knowledge of their application. The presentation of the subject in this book takes up so much space and deals at length with so many problems, that at first sight it does not seem that much progress had been made towards simplifying the subject. But it must be borne in mind

that before fundamental principles can be recognized a wide outlook must be made over the whole of medicine, and as this book is concerned not only with the enunciation of these principles but with the evidence of their existence a great many details had to be used.

In addition, it is a matter common to the history of all sciences that when the laws or principles governing the production of phenomena are first recognized it takes a long time before they are sufficiently understood to be profitably employed. In such a subject as medicine a preliminary knowledge of the structures and their functions which regulate and control the activity of organs is required. In many cases that kind of knowledge is so defective that much preliminary work has to be done before the principles can be applied.

This fact has been recognized, and the reader will find many instances in which the principle cannot be convincingly employed. Thus in dealing with heart block, the structures and functions of the genetic or conducting system are still so imperfectly known that it is possible to see only dimly how the principles should be applied. Recognizing this, details are given of individual cases many of which are incapable of being interpreted for the reason just stated.

In Chapter XIII. is given a classification of symptoms. It is felt that when the significance of this classification is grasped it will greatly help towards a simplification of medicine, inasmuch as it affords a means by which the phenomena perceived in the different departments of medicine can be correlated. The value of this classification is illustrated in dealing with irregular action of the heart.

There is every reason to anticipate a time when the principles of symptomatology will be so well understood that it will be possible to recognize the symptoms produced by all organs when a full knowledge is acquired of the nature of the symptoms in one. Once this idea is grasped, then by concentrating upon the unravelling of the symptoms of the simplest and most easily observed organs a knowledge of the more complex organs and those difficult of approach will be acquired.

To achieve this purpose it will not be necessary to search for all the symptoms present in a sick individual—only those few which reveal the nature of his ill-health. These, in all likelihood, will often be found to be those which can be recognized by the unaided but trained senses of the doctor. By this means the benefits of medical progress will be rendered available to patients under all sets of circumstances.

CHAPTER I

A Review of Modern Cardiology

In order to bring before the reader the grounds on which it is desired to obtain a better conception of medical research I give here in brief outline a few of the experiences which forced the defects of medical knowledge on my attention.

A few years after entering general practice I realized that I did not understand the nature of any of the signs of ill-health presented by my patients, not even in those patients where my treatment seemed to be successful. I turned to textbooks for enlightenment, but found in them no help. It was thus forced upon me that the knowledge of medicine had not advanced far enough to enable an understanding to be obtained of the nature of the symptoms of disease. It became evident that if medicine was to be practised intelligently it was essential that this kind of knowledge should be attained. Ignorant though I was of the methods and principles of research, I nevertheless determined to make an attempt to improve my knowledge of this matter.

It is more than forty years ago since I came to this determination, and I have continued the investigation ever since. I recognized at the outset that I must have a definite object, and I formulated two objects which I set out to achieve. The first object was to find out the factors concerned in the production of symptoms, and the second was to know what bearing the cause of the symptom had on the patient's future health—in other words, the prognostic significance of symptoms. It had become abundantly evident to me as a doctor dealing with diseases common amongst the people, that without this latter kind of knowledge the practice of medicine could never be intelligently pursued.

For several years my endeavours yielded no fruit, for I did not know how to set about the enterprise. Practically, research in medicine then, as now, was haphazard and guided by no sound principle. Gradually, however, there emerged a conception, at first vague, which became clearer, until I was able to formulate a certain kind of principle. One of the subjects I had taken up was the nature of the irregular pulse. In contemplating a sphygmogram of an irregular pulse the question arose, What was it that this sphygmogram revealed? It was realized that it gave evidence only of the activity of the left ventricle. The next question arose, What were the other chambers of the heart doing?

At that time, about 1890, the movements of the different chambers of the

heart were very imperfectly understood. The belief was widely held that the two sides of the heart could contract independently of one another (hemi-systole). The recognition of the activity of the auricles in clinical medicine was practically limited to the murmur caused by the contraction of the left auricle in mitral stenosis. After I had devised a simple method by which records of three and sometimes of four chambers of the heart could be obtained, I employed it for the interpretation of irregular heart action. I had already recognized that the evidences of hemi-systole were inconclusive, and the problem had resolved itself into the question of what the auricles were doing when the ventricles acted irregularly ?

This is the principle which I adopted at that time, and it is the one which guided me for many years. It may be pointed out that it is this simple principle which has been the guide in all researches into irregular heart action up to the present day. Because of its simplicity the importance of this principle is not grasped even by many who use it.

The first form of irregularity to which I applied this method was the ' intermittent ' pulse, and the records of the radial and jugular pulse showed that the ventricle contracted earlier than and independent of the auricle—the latter maintaining its regular rhythm. This is now recognized as the ventricular extra-systole. This was in 1892. Although I called the attention of physicians and physiologists to this fact, not one of them would accept my interpretation and not one of them appreciated the method employed.

It was only some years later, when experiments on the frog's heart and the dog's heart had shown that my observation was correct that the value of the method began to be recognized. Since that time many others have participated in this investigation by the same and other means, and a great many instructive facts have been brought to light.

During these years when engaged in this investigation the other object of my enterprise was never lost sight of, *i.e.* the bearing of the cause of the symptom on the patient's future health. I could see no other way to obtain this knowledge than to watch individual patients year by year to see what happened to them. There seemed to be no principle beyond this which would serve as a guide.

In this endeavour to find out the significance of heart signs, it gradually dawned upon me that the essential matter in every case was a question of heart failure. Did the signs indicate heart failure, or foreshadow its oncoming ?

This, then, directed my inquiries into the nature of heart failure, and it became more and more evident that the object of all inquiries into the heart in clinical medicine had for its object the question of the heart's ability to maintain an efficient circulation, and that without a knowledge of heart failure all such inquiries would be rendered futile.

The significance of this fact will be realized if we contemplate two great enterprises that have been undertaken by large numbers of physicians during the past century. It is about one hundred years since auscultation was applied to the heart. Shortly after its introduction the profession at large

became deeply interested, and for the next fifty years, nay even up till now, the sounds of the heart have been the subject of constant inquiry. When at this date we look back and review the effects of this inquiry upon the practice of medicine we can recognize that the exalted expectations which were formed have not been fulfilled and that it can now be seen that auscultation can throw but a little light upon the essential problem of heart failure.

The question of irregular action of the heart may be said to have begun with Marey's experiments upon the frog's heart some sixty or seventy years ago. During the last thirty years the clinical study of irregularities has proceeded apace, and we see here how closely history has repeated itself, for it will be found that for the practice of medicine the study of the irregular action of the heart has been of even less profit than that of the sounds of the heart. In both instances, no doubt, valuable facts have been revealed, but they occupy but a small portion of the field of cardiology, and so far, in place of helping to a knowledge of the essential matter of heart failure, they have actually obscured the subject and misdirected investigations into profitless paths.

How little is known about heart failure can be found by any one who seeks for information in recent writings of cardiologists. It is perfectly evident that to obtain a knowledge of heart failure a far more intimate knowledge of the manner in which the heart performs its work must be acquired, and that the conceptions which have guided investigations so far are unsuited to carry the matter far enough.

In the movements of the heart, structures and processes are involved of which the knowledge is so vague and indefinite that it is not possible to recognize those that take part in some simple manifestation, such as the increase in rate in response to effort or the production of an extra systole. Indeed, if the matter be looked into it will be found that it is not possible to interpret any reaction of the heart, whether experimental or clinical, with that particularity necessary for the solution of medical problems. Such explanations as are given in physiological or clinical textbooks are imperfect, meaningless, or misleading.

The reason for this is not far to seek. The explanations given are based on conceptions of the heart-beat obtained at a time when very little was known about the matter. The modern notions of the heart-beat began with experiments some sixty or seventy years ago. During these early experiments it was found that the heart responded readily to electrical stimulation applied directly or through stimulation of the sympathetic or vagus nerves. The reactions that were obtained were interpreted according to the limited knowledge possessed by the investigators.

Since those days what is called the 'conducting system' has been discovered, and the investigation of this system reveals that it plays an important part in every movement of the heart, so that all interpretations which do not take cognizance of this structure are bound to be incomplete.

Endeavours have been made to study the function of the conducting

system, but with little success. The reason for this is, that the manner in which it performs its functions is different from that of any other structure which has hitherto been investigated, consequently the conceptions of vital activities which are employed to-day to explain physiological reactions are not sufficient to supply this information; moreover, experimental methods are inadequate to reveal the peculiar features of the functions of this structure.

For the proper understanding, then, of the heart's movement a new field of inquiry has to be explored, new conceptions have to be found to interpret the phenomena, and suitable opportunities have to be sought for its investigation. These opportunities are to be found mainly in clinical medicine.

The facility with which reactions can be obtained by the use of electricity as a stimulant has led to the employment of a method which yields phenomena markedly different from those which occur in nature and which are observed by the clinical investigator. The failure to recognize this difference has led to the interpretation of natural reactions in the light of these artificial reactions, and this has added another obstacle to the understanding of the manner in which the heart performs its work.

The conception held to-day of the effect of sympathetic and vagal activity upon the heart, as well as that of the law of 'all or nothing', which for over fifty years has hung like a millstone around the neck of investigators, and the so-called functions of heart-muscle fibres described by Gaskell and Engelmann, were all based on the interpretation of reactions obtained by electrical stimulation at a time when the part played by the conducting or genetic system was unsuspected.

This reference to pioneer investigators is not intended to cast any reflection upon them. All investigators are ignorant, otherwise they would not be investigators, for it is the consciousness of ignorance which leads a person to investigate. The new facts brought to light cannot be satisfactorily explained at first, but in order to guide him in his pursuit the investigator gives an explanation which he recognizes as provisional. He then sets out to find other facts which may support his explanation or provide him with a better one. Thus all investigators can give at the early stages only provisional explanations, which are dependent upon the state of knowledge at the time. A great pioneer like Gaskell did not look upon his interpretations as settled beliefs, and he was ever changing his point of view. I heard a distinguished physiologist once reflect upon Gaskell because of his habit of changing his views on matters that he was investigating. Gaskell recognized that his description of cardiac muscle functions was but provisional.

The acceptation of such provisional explanations given by pioneer investigators as settled beliefs is one of the potent causes which has hindered the progress of cardiology. The absence of a knowledge of the part played by the various structures and processes concerned in the heart-beat has rendered many schemes of investigation abortive. Indeed, such a result might have been predicted from the time of their inception.

Many illustrations of abortive enterprises could be cited, but in recent years

there has been one which shows how the conception of research at present held failed at a most important juncture.

For many years it has been accepted that it was well known how to estimate the physical fitness of individuals. Not only were experienced physicians considered qualified to assess the insurance value of lives, but recent graduates were supposed to be able to determine the fitness of recruits for the army and the navy.

When the War broke out a great responsibility was thrown upon the medical profession in that they had to select the men fit for military service. When thus called upon they were found not to possess the required knowledge. Large numbers of perfectly healthy individuals were rejected because of the presence of some sign whose value the doctors were never taught to assess. Unfit men were accepted because no physical sign of disease was detected. So glaring and so numerous were the mistakes, made not only by doctors unused to such duties but by the military authorities themselves, that steps were hurriedly taken to supply more accurate information, but in vain. Equally glaring defects in knowledge were revealed when the health of the soldiers began to give way. Within a year of the outbreak of war tens of thousands were invalided and lay in hospitals, being treated for supposed affections of the heart. Many were not ill, but others were ailing; but as no doctor could tell the nature of the complaints, the ill-health was put down to the heart, because a murmur was present, or because the heart seemed unduly excitable, or exhibited an innocent form of irregularity; and thus arose those diagnoses of V.D.H. (Valvular Disease of the Heart) and D.A.H. (Disordered Action of the Heart), the stand-by of the puzzled doctor during the War.

In several countries engaged in the War strenuous attempts were made to find a reliable method for estimating the fitness of individuals and for understanding the nature of the ill-health described as the 'soldier's heart'. Hospitals for the investigation of the heart were set apart; groups of doctors with special knowledge of the heart were detailed for this investigation, and every conceivable device was employed. For several years such investigations were pursued with the greatest industry, and if to-day we seek for the increased knowledge of the heart which has accrued, it will not be possible to detect the slightest gain.

The failure to obtain a satisfactory method for estimating the physical fitness of individuals was recognized, and since the War the matter is still in course of investigation with little prospect of success so long as the present conception of investigation prevails.

During the War I was deeply interested in these matters and spent a great deal of time in similar attempts. Like my colleagues, I failed to make any progress. I had recognized that in over 90 per cent of the cases of 'soldier's heart' the heart was not in any sense the cause of the ill-health, the heart symptoms being but a part of the phenomena of ill-health.

During this inquiry I became convinced of the possibility of a conception which for years I had dimly perceived. Many of the symptoms in a sick man

could be referred to a common cause, and the idea arose that the production of symptoms was governed by definite laws, and that if these were recognized it would be possible to classify symptoms according to their affinity in nature. If such a classification could be obtained it would render easy of comprehension that chaotic mass of symptoms which hampered rather than helped the progress and practice of medicine. It seemed possible that in such a classification some principle might be found which would do for medicine what the atomic theory had done for chemistry.

On a review of the various forms and aims of medical research then in vogue, not one was found devoted to the phenomena of ill-health. The failures, to which allusion has been made, could be attributed to a lack of knowledge of such phenomena, and it was evident that such a matter called for a specific and systematic investigation.

I therefore gave up practice to devote myself to this task and started an Institute for Clinical Research in the university town of St. Andrews. Recognizing my incapacity for such an undertaking, I invited the general practitioners of the town and certain of the professors and teachers in the university to join me in the quest.

During the past five years we have strenuously pursued an inquiry into the nature of ill-health. Much difficulty was experienced owing to the absence of knowledge of the manner in which the functions of organs were regulated and carried on. Such descriptions as were given in the usual textbooks failed to provide the knowledge with that particularity which was necessary for our purpose. Much time had therefore to be spent in getting a better understanding of how organs normally functioned. In course of time there gradually emerged certain principles which, if they do not fulfil the expectations with which the inquiry was started, have carried the matter of symptomatology a long way forward.

A method of investigation has been evolved, based upon these principles, which enables research to be carried on in a systematic manner in place of the haphazard methods hitherto pursued. An insight has been gained into the structures and processes concerned in vital activities which enables the nature of symptoms and other reactions to be recognized. A classification of symptoms has been obtained based upon the only sound principle in science, *i.e.* on the affinity in nature of the varied phenomena.

The application of these results to the understanding of the phenomena of ill-health has thrown an unexpected light upon many obscure problems, and there is good reason to look forward to a continuous progress, as it becomes better understood how to apply this line of investigation.

In this book there is described the application of this method to the ill-health which results from affections of the heart. If the results herein described do not seem to have carried the matter far, this is because the subject is at a very early stage and a great deal of time has had to be spent, not only in presenting the new conception but in demonstrating how completely the old conception had failed. In this attempt to replace an imperfect conception by

a better the greatest difficulty has been experienced, for men do not easily give up deeply cherished beliefs.

There are many aspects of abnormal heart action which cannot yet be comprehended. I have, therefore, given in some detail a brief history of a number of cases, in order that the reader may test the truth of the point of view put forward, and also to record facts which cannot yet be interpreted. The reason for this is that I recognize that the present description can only be provisional, and I am anxious to provide data which can be used to confirm these views or to modify them, or to supersede them. If I have succeeded in demonstrating the inadequacy of the present views my purpose will have been fulfilled, for then those who realize this inadequacy may be stimulated to seek for an adequate explanation and give the views here enunciated consideration. If these are found wanting, others more satisfactory will have to be sought for, and thus progress will become assured.

CHAPTER II

The Significance of Symptoms

Diagnosis.
The object of a prognosis.
Prognosis and treatment.
Modern medicine and prognosis.
Prognosis in the past.
The effects of unproved beliefs.

Diagnosis.—Notwithstanding the great amount of consideration that has been given to diagnosis and prognosis, there still exists so much confusion that their respective places in medicine are not clearly recognized. The term diagnosis implies the discrimination of diseases by their distinctive marks or symptoms. To make a diagnosis is assumed to be one of the chief duties of the doctor, as it is commonly believed to form the basis of medicine and to give the indication for treatment. The importance of diagnosis has bulked so largely in the minds of the profession that attention has been concentrated less on improving clinical methods than on superseding them by what is called laboratory methods, under the belief that these are more reliable, and because they reveal more symptoms. So long as the belief is held that diagnosis forms the basis of medicine and affords an indication for treatment, the heaping up of a mass of symptoms will continue.

It is instructive to look at some aspects of diagnosis. If we take as an instance pneumonia—a condition in which the ill-health has been the subject of long and painstaking observation in both the hospital and laboratory—we will get some inkling as to the part diagnosis plays in medicine. The term pneumonia implies an affection of the lungs, but the ill-health which is present or the death which may result is not due to the lung condition. The lung affection is only one of a great many phenomena which are present when a man is ill from pneumonia. It is not even one of the disturbances which imperils life. The detection of a microbe which provokes the ill-health throws no light upon the conditions which made the man ill and which may lead to death.

Even when a laboratory diagnosis is made we are still in the same plight. Such a diagnosis may tell us that the patient has got typhoid fever, but this gives us no information as to the danger, or absence of danger, or where we are to look for the danger. In the same way an electrocardiogram may reveal

an abnormal heart action, but it throws no light upon the capacity of the heart to perform its work, which is the essential matter to know.

By auscultation a murmur may be detected and accurately 'diagnosed', and the patient may be said quite correctly to have mitral regurgitation, but that throws no light upon the condition of heart failure from which he may be suffering. In the same way a correct diagnosis of an extra systole is equally lacking in information as to the reason of heart failure, should it be present. A man may have a cough and expectoration ; the diagnosis of bronchitis may be correct, but that does not account for the illness from which he may suffer. The vast majority of diagnoses made by the most skilled are often incomplete and, in consequence, misleading.

When a diagnosis is taken to indicate a line of treatment, the treatment is invariably of the 'rule-of-thumb' kind. A laboratory diagnosis may confirm a clinical diagnosis of diphtheria. This is taken as an indication for the administration of anti-toxin serum, and while some who get it obtain benefit, a great many get it who do not need it, while others get it who derive no benefit. For this latter class practically nothing more is done, and indeed the faith in the anti-toxin treatment prevents anything more being done for them.

A diagnosis is made of appendicitis, and many surgeons take the diagnosis as an indication for operation, with the result that a great many people have their appendixes removed unnecessarily, and their lives placed in jeopardy. In such an instance surgery has only progressed so far as to make a diagnosis, but has not gone so far as to obtain a prognosis.

The object of a prognosis.—Medicine is essentially a utilitarian subject, having for its purpose the combating the ravages of disease.

When a doctor examines a patient the essential question in every case is, 'What is to be the outcome of his complaint?' As we have seen, diagnosis can rarely afford this kind of information. The symptoms that are suitable for diagnosis are seldom available for prognosis. The symptoms by which we can tell that a patient has scarlet fever or measles are not those which tell us whether the patient is to do well or ill. The reason for this is that the diagnosis is usually based upon the symptom or sign which happens to impress the examiner, and these symptoms may be produced by organs which do not reveal the phenomena which tell us whether there is danger or not.

Prognosis and treatment.—In addition to affording valuable information of the presence or absence of danger, prognosis requires that the source of danger, if present, should be known, so that it might be possible to combat it. It is the purpose of treatment to combat danger, and if it is to be intelligently directed the source of danger and its nature must be recognized.

This requires a knowledge of the structures and vital processes concerned in the production of ill-health. With their recognition we obtain a definite object to which treatment should be directed.

If ever treatment is to become scientific and to escape from empiricism, the principles of prognosis must first be understood and employed in the examination of patients.

Modern medicine and prognosis.—The distinction drawn between diagnosis and prognosis seems so clear that it might be thought unnecessary to insist upon the importance of prognosis, but when the lines along which modern medicine is developing are considered it will be found that prognosis is being ignored. In place of looking upon it as the highest level to which scientific medicine can aspire, the tendency is to practise medicine and to administer treatment without considering the prognosis. This is particularly the case with those who devote themselves to the bacteriological and laboratory side of medicine. Treatment by vaccines and sera is given according to laboratory findings, and not according to the condition of the patient. Books on modern therapy or vaccine therapy do not even mention the word prognosis. Surgery is tending the same way. If a patient is ill and something is wrong which an X-ray examination may reveal, an operation is undertaken without considering the question whether the condition revealed is the cause of ill-health. Perusal of modern surgical textbooks throws no light upon prognosis ; usually the subject is barely mentioned.

Prognosis in the past.—It may seem uncalled for to dwell upon the importance of prognosis, for many authors and teachers pay attention to it, and it is also true that for many centuries the importance of prognosis has been recognized. It is also true that many physicians and surgeons and general practitioners, after long experience, have acquired a remarkable knowledge of prognosis, but where the non-progressive, if not retrogressive, character of this kind of knowledge lies is that the knowledge is a matter of individual experience, and those who had got the knowledge were not able to communicate it to others, for the reason that they themselves could not explain why they assessed the value of the symptoms, except as being based upon their own experience. As one individual cannot fully enter into the experiences of another, such knowledge could not be conveyed except as a matter of faith. One result is that the young doctor did not begin where the old one left off, for the knowledge departed when the old one died, and the young doctor had to begin where the old one started. It is this Sisyphean form of activity which renders medicine in this very important department unprogressive.

The effects of unproved beliefs.—So completely has modern medicine failed to realize the importance of prognosis that it is necessary to draw attention to some of the consequences of this defect in knowledge of a subject so essential to the rational practice of medicine.

It is customary to speak of education as a torch which sheds light in dark places, and which is handed down by teachers from generation to generation. Accepting such a simile, one must remember that light casts a shadow. In the course of this torch-carrying through the centuries, the light of knowledge has been accompanied by the shadow of superstition. That this should be so but illustrates a characteristic of the human mind. Medicine has been taught for centuries, and the teachers of to-day are directly descended from those of the dark ages. The teacher in medicine takes up his duties at a period of life when he has had little experience, and no opportunity of testing the truth of what

he teaches. He is therefore bound to tell the students what he himself had been told. Once a man begins to repeat a thing he gradually comes to believe it, and after a few years the teacher believes implicitly his own teaching. It is for that reason that so many teachers are slow to appreciate a new conception, especially when it casts doubt upon what they have been teaching for years.

In some remote past the idea arose that a heart to be normal must be regular in its rhythm, and that any irregularity must be looked upon with suspicion as a possible sign of gravity. A few years ago, when I was present at a meeting of medical examiners for life insurance—a body reckoned to consist of specialists on prognosis—I was struck by their attitude towards irregular heart action. Experienced physicians stated that they systematically penalized or rejected candidates if the heart showed any form of irregularity. It seemed incredible to them that a healthy heart could be irregular, while an innocent abnormality like an extra systole was regarded as justifying rejection. Even to-day there are heart specialists who consider the physiological irregularity of the young, or an extra systole, an indication for treatment and for limiting the efforts of the individual. Many physicians who in the abstract will admit that irregularities may be of little or no significance, invariably place restrictions upon the individual patient and subject him to treatment. I have seen so many healthy people treated in this way that I am compelled to call attention to the urgent need for a better understanding of heart irregularities.

Humiliating as is the ignorance of the profession towards irregularities, still more humiliating is their attitude towards murmurs and modified sounds. While we justly acclaim Laennec for his discovery of auscultation, it must be admitted that its introduction into medicine has not only for a hundred years hampered the progress of knowledge of heart affections, but has done more harm than good in that many people have had the tenor of their lives altered, have been forbidden to undertake duties for which they were perfectly competent, and have been subjected to unnecessary treatment. This is not due to auscultation as such, but to the inability of the profession to assess the value of the phenomena which auscultation revealed. Before it was known what produced the sounds of the heart, modifications were taken as signs of evil significance, and that misjudgment to-day does incalculable evil to the practice of medicine.

The position of prognosis to-day cannot be better expressed than in the words of the great physician, Sir William Gairdner, who declared, in a discussion on the insurance of patients with heart affections, 'Whether any data can be procured that will enable our successors to deal with cases of extra risk, we have none, so deal with them either by loading or rejecting, or shutting our eyes'. He goes on to say that the life insurance examiner 'deals with his cases in the most curiously unsatisfactory way—either by rejecting the life altogether, or by putting on a big loading, which it must be admitted we assess upon no very sound or clear principle, or, on the other hand, by winking at the defect and passing it over'.[1]

[1] *British Medical Journal*, 1898, vol. ii. p. 758.

When we reflect that for all these years prognosis has been the chief duty of life insurance examiners, and the methods which are indicated in the above statement are pursued to-day, as one can see by reading any of the modern books dealing with prognosis and life insurance, the call is urgent for a better understanding of this matter.

CHAPTER III

The Principles of Prognosis

The neglect of prognosis.
The importance of the trained senses.
Research into prognosis.
A new method for investigating prognosis.
Sources of prognostic symptoms in heart affections.

The neglect of prognosis.—I am at a loss to know how to present the subject of prognosis in a manner that will bring home to the reader how essential prognosis is for the intelligent practice of medicine, and how its persistent neglect in recent years not only hampers the progress of medicine, but has so led to the diversion of medicine from the path of progress that in some respects medicine is actually going back. The years I spent in general practice impressed its importance upon me, and I view the matter in an aspect different from that of men whose experiences have been restricted to laboratory or hospital and consulting practice. In discussions with such men I have been astonished to find how differently we view the matter. To them prognosis is so simple that it is easily acquired and easily taught, so that young men fresh from the schools are considered competent to examine people for life insurance and recruits for the army.

On the other hand, I see the matter as one of the most difficult in medicine. It represents the highest development of medicine, all other departments being concerned in acquiring a knowledge of a necessary but rudimentary kind, which is but preliminary to acquiring a knowledge of prognosis. The physical signs used for diagnosis, whether detected by the senses or by laboratory methods and mechanical devices, stand in relation to medicine just as the simple rules of arithmetic do to the higher mathematics.

In order to bring my point of view home to the reader I am unfortunately compelled to introduce more of my personal experiences than may seem desirable, but I cannot see my way to make my points clear in any other way. It is impossible for me to convey to the reader the great numbers of healthy people who have been rendered miserable, who have been condemned to a life of restriction, have been subjected to all forms of treatment, some of which have imperilled their lives, not by doctors in general practice, but by leaders of the profession and teachers of schools. I have also seen great numbers of people with some slight impairment who have been made miserable chronic

invalids by a gloomy and unjust prognosis. I therefore write this under a sense of grave responsibility, and urge that the following descriptions should receive thoughtful consideration, and the ideas here set forth tested, and strenuous efforts made to supersede them if they are found wanting.

In setting forth this subject I shall present it, if possible, in such a way that the reader may see the reasons for the backward state of prognosis and shall indicate the steps taken to provide this knowledge ; I shall endeavour to show that there is gradually coming to light a recognition of the fundamental principles on which research on prognosis should be conducted. In subsequent chapters it will be shown that the matter has been carried so far as to hold out the prospect that an inquiry upon these lines will add greatly to our knowledge not only of prognosis but of every department of medicine.

The importance of the trained senses.—I have referred to the fact that experienced doctors acquire a knowledge of prognosis which at times is so accurate that it seems almost miraculous to the uninitiated. Perhaps there is nothing that more astonishes the young doctor who has been trained to examine patients by means of a great variety of laboratory devices than to find how helpless he is when away from these devices, and to discover how a trained practitioner may speedily arrive at a diagnosis in cases which puzzle him. But more important than diagnosis is the manner in which the trained and experienced practitioner can tell whether the patient is in danger or not. In my young days I was profoundly impressed with the rapidity and certainty with which diagnosis and prognosis were arrived at by experienced physicians and general practitioners, and I set myself to find out the grounds on which they based their opinions. In the course of inquiry into this matter I realised that it was an outcome of prolonged study of individual cases and of the phenomena of ill-health which could be perceived only by the trained senses of the observer. They had recognized certain phenomena and had associated these phenomena, some of them with grave conditions, and some of them with conditions of minor importance. I remember being greatly impressed by an experienced physician in a consultation over a patient, an elderly gentleman, suffering from extreme heart failure in whom I feared that death was impending. The consultant, after a brief and seemingly superficial examination, cheered the patient greatly by telling him that there was no danger at present, and that he would get a great deal better. I asked him on what grounds he gave such a favourable prognosis, and he replied that patients with that kind of heart failure never died in a first attack. As years went on I realized the truth of this statement, but it took me many years to understand the reasons for it.

In the same way an experienced doctor can tell in a few minutes, for example, whether a woman with a fever during her puerperium is in danger or not ; whether a patient with typhoid fever or other infection, even where a diagnosis cannot be made, is or is not in danger. The reason for this I also set out to find.

This kind of knowledge cannot be over-estimated. The patient and his friends are indifferent to the most learned diagnosis, but they are deeply interested in the question of danger or no danger, and they are right. They all

know that if there is danger the source of danger should be recognized, otherwise it cannot be combated.

The steady pursuit of this kind of knowledge enables one now to recognize in the vast majority of people with affections of the heart whether they are in danger or not, by methods as simple as those of the experienced doctor to whom reference has just been made. Time and again I have seen patients submitted to prolonged examinations by every conceivable means, not only by the individual physician but by groups of specialists who have had the patient for days under examination without their being able to recognize the nature of the complaint, though it was made perfectly clear by a few minutes' examination by using the trained senses.

The methods employed by the use of the trained senses are at the present day considered to be ' unscientific ' and less valuable than the methods which require laboratory devices. The reverse is the case. Laboratory devices in medicine represent an elementary stage of development. The highest development requires the value of the symptom to be assessed, and to be able to do this, the matter has to be carried far beyond the reach of laboratory methods.

Research into prognosis.—It is more than forty years since I set out systematically to investigate the mechanism of symptoms and their prognostic significance. The former object necessitated the employment of mechanical devices, and it was comparatively easy to make some progress in this manner of research. On the other hand, it was extremely difficult to find a method which could be employed for research into prognosis.

In regard to heart cases I collected carefully a number of symptoms and tried to differentiate one symptom from others which it resembled. Then I proceeded to follow individual cases to watch any modification of the symptom and to see if its cause had any relation to increasing ill-health or to the improvement of health. In this way a great number of records of murmurs and modified sounds of the heart, of variations of rate and rhythm, was obtained, but little progress was made. After some years I reviewed my work and was disheartened with my failures. I then put to myself the question, ' What is there to be afraid of ? ' and the answer ' heart failure ' immediately gave me a clue and I set out to discover how heart failure was brought about and the symptoms that reveal its presence. This has been the main object of my researches for the past thirty years.

I thought until recently that the only method was by following the patients to see what happened. This is the method that I have pursued, and it has yielded a good deal of very useful information. It was long and tedious in many instances, for I had to watch individual patients often from ten to fifteen years or longer before I could be sure of the significance of the symptom. I had to see the patients undergo periods of stress from over-work or intercurrent illnesses.

Some of my results have been accepted by the profession, others have not. Some of those that have been accepted are recorded in text-books on the heart as if they were matters so well known that it is unnecessary for the

author to indicate whence he got his knowledge, or to give any description of how the knowledge was obtained.

As an illustration take the reference to that condition of irregular action of the heart which I have called the 'youthful type', as it is very frequent in the young, also recognized as a 'sinus arrhythmia'. This irregularity is now described as having no prognostic significance, but it does not seem to occur to any author to inquire how he has obtained this knowledge. I do not doubt he would say it was learned from his own experience, and would not admit that he had borrowed it from me, or recognize at what expense of time and trouble it had been acquired.

It took me fifteen to twenty years of patient observation and it required the collection of an enormous number of records before I felt confident in the soundness of this prognosis. I had had the opportunity of seeing people in childhood, and observed their hearts during various illnesses. I watched children grow up and pass into manhood and womanhood, and observed how they bore themselves during periods of stress in playing games and undertaking hard work. The symptoms observed had to be compared with those in people with failing hearts, and after many years' patient labour this prognosis was established.

When I removed to London in 1908 the first patient I saw in consultation was a girl who had fainted and to whom a consultant had been called in. He found the pulse irregular, and had placed her in a nursing home to undergo Nauheim treatment. She had been three months in bed, and the heart was still irregular, the irregularity being of the youthful type. Shortly after this a boy was brought to me from one of the public schools. For a year he had been forbidden to play games. The story was that about two years before, after a febrile illness, his heart was found to be very irregular. He was seen by a London consultant, who put him to bed and treated him for three months. At the end of this time the pulse was still irregular, and the parents took him to a city on the Continent where he was again treated for several months. As there was no improvement he was taken to another renowned authority in another city, who treated him by hypodermic injections with various drugs, and after several months, the heart being still irregular, the sorrowing parents were informed that the boy had an incurable disease, and that he would never be able to play games or undertake any strenuous physical work. I found the irregularity to be that of the youthful type, and recommended that the boy should at once begin to play games and lead the life of a healthy youth. This he did, and after his schooling was over he entered the army, and went through the War with no trouble from his heart. I quote this case to show the state of knowledge abroad as well as at home.

I could cite innumerable similar instances, and if any one cares to read the literature on the subject before it was dealt with in the first edition of this book, he will rarely find any reference to the subject, and what references there are will be forebodings indicating that such irregular hearts are an object of grave suspicion.

One reason for citing these experiences is to show how the importance of this line of investigation has never been appreciated, and is not yet appreciated. Another reason is that if some remedy had been discovered which would have 'cured' this condition the whole medical world would have resounded with its praise; but because a far more important discovery was made, by the employment of methods which belong to the higher sphere of medicine, the value of the discovery is unappreciated.

A new method for investigating prognosis.—I had applied the same line of investigation to discover the prognostic significance of other signs or symptoms exhibited by the heart, and the results were so valuable that I for long thought that it was the only method by which the information could be acquired. But since we began to apply the principle of the 'reflex arc' a new conception has enabled me to see that the same kind of information can be acquired by much less tedious and less prolonged methods of investigation. This has come about through the development of the idea that amongst a multitude of symptoms a sick man may exhibit, only a few of these are essential for prognosis. In the description of the manner of death it will be shown that heart failure is brought about by interference with the functional efficiency of the muscle. The heart may provoke a great many symptoms, many of which give no information of this sort. It is necessary, then, to find out the symptoms which give a knowledge of the functional inefficiency of the heart muscle, so as to separate them from those symptoms which do not afford this information. The present state of knowledge of affections of the heart does not permit this information to be acquired, and consequently the principles which should guide prognosis cannot be applied.

Sources of prognostic symptoms in heart affections.—The following is briefly the line of reasoning which is employed to get this knowledge and which will be developed and elaborated.

There are three main structures in the heart which give rise to symptoms. These are, first, the conducting system, or what I shall in future call the 'genetic system', for reasons which will be given later. This system is concerned in the production of every movement of the heart. Impairment of this structure is only of importance in so far as it interferes with the functions of the heart muscle. Therefore all variations in the rate and rhythm of the heart are without prognostic significance unless they embarrass the heart muscle.

A second structure which gives rise to symptoms are the valves of the heart. Here, again, any damage to them is only of importance if it embarrasses the muscle in its work.

The third structure is the muscle, and the inquiry resolves itself into the integrity of the muscle and the state of its efficiency.

These are briefly the considerations on which the principle of investigating prognosis should be based. Each symptom that is detected should be carefully examined to find out the structures involved in its production. When these are recognized, the question arises—Does the health or the life of the patient depend upon the integrity of this structure? This is the object to which

all inquiries should be directed when the value of a symptom is being assessed.

The unfortunate matter is that medical knowledge has not advanced far enough to recognize the structures which are concerned in the production of a single symptom. This defect in knowledge has involved the search for some sound principle which will guide medical investigations to remedy this defect. Every department of medicine will remain backward and unprogressive until this kind of knowledge is acquired. It is manifestly the kind of knowledge which tells us if there be danger, and where the danger lies. When we know this we are then in a position to attack the problem of combating the danger with a clearness of perception hitherto unattainable. It will thus be realized that it is only by this kind of knowledge that treatment can be rescued from that empiricism which dominates every department of practical medicine.

CHAPTER IV

The Principles of Prognosis (*continued*)

The reflex arc and prognosis.
Prognosis and the multitude of symptoms.
The kind of prognosis wanted.
Prognosis and heart failure.
The manner of death.

The reflex arc and prognosis.—In the course of an investigation into prognosis at our Institute at St. Andrews there gradually emerged certain principles which put the whole question of symptomatology in a new light. The first step was the recognition that symptoms were due to disturbances of those processes and structures which are concerned in regulating the function of organs. As these processes are spoken of as reflex processes, we put forth the theory that symptoms were due to the disturbances of the normal reflexes. In our investigations we applied what we called the Principle of the Reflex Arc, which will be discussed later. This principle necessitates the recognition of the structures which regulate the activity of organs, with an analysis of the arc into its separate elements and their individual function, so that the symptom could be referred to the element which was disturbed. In this way we gradually were able to recognize the origin and nature of many symptoms, and there emerged a method by which we could classify symptoms on principles based not only upon their affinity in nature, but also upon their prognostic significance.

Prognosis and the multitude of symptoms.—The principle upon which these classifications are based is that in a sick man many organs may be disturbed and each organ gives rise to one or more symptoms. One sick person may therefore show a great many symptoms. Such symptoms differ widely in their diagnostic and prognostic value. The difference in prognostic value depends upon the part played in the bodily economy of the patient by the organ whose disturbances gives rise to the symptom. Some organs are essential to the maintenance of life ; others play a more or less subsidiary part. Disturbances of the former may put life in jeopardy. Disturbances of the latter may cause little or no interference with the vitality of the body. Some symptoms may be prominent, although of little value in helping to a prognosis, while others, subtle and obscure, may be of the greatest value.

Symptoms have therefore to be considered, not only from the point of view of the organ or structure producing them (diagnosis), but also whether

they indicate the presence of conditions which interfere with those structures and processes on which health and life depend (prognosis).

The recognition of this fact, that the value of symptoms depends upon the function of the structure concerned in their production, provided a new line of investigation, viz. to find out the structures which are concerned in regulating the function of an organ, and the part that each structure played in this regulation. Unfortunately, the knowledge for such a purpose is at present far too scanty to carry the matter far. In the heart, however, there has been a considerable advance in our knowledge of the process and structures concerned in the heart-beat and its regulation. Each of these structures plays its particular part, and the disturbance of each has its own peculiar significance. Their value is determined by the purpose we have in view. If it is to obtain a knowledge of the conditions that influence the rate and rhythm of the heart one class of symptoms has a particular value. If it is to understand the capacity of the heart to continue to maintain an efficient circulation, then another kind of symptom must be sought for.

The kind of prognosis wanted.—In seeking for a line which will guide to a prognosis it is necessary first to understand clearly the knowledge that is wanted. It is manifestly a different kind of knowledge wanted for a prognosis in a patient with a broken leg, in another with a gastric ulcer, in another with a pneumonia, in another with a heart affection.

The object of the prognosis will probably be found in the answer to the question, 'What is there to fear?' In the conditions just mentioned the answer will be different in each case. This book is concerned with the last of these conditions, and the question to be answered on the examination of a patient with a heart affection or a supposed heart affection is, 'What is there to fear?' The fear is that the heart should fail.

Prognosis and heart failure.—When the matter is considered it will be found that it is the fear of heart failure which influences the patient who imagines there is something the matter with the heart, to consult a doctor. It forms the object of the doctor's examination, and it is the reason for the employment of the many methods now considered necessary for the examination of the heart. Whenever a symptom is detected the question arises, 'Does the symptom indicate the presence of heart failure, or does it foreshadow its occurrence?' It is the fear of heart failure which not only forces the patient to consult his doctor but makes him willing to submit to all forms of restriction and treatment.

In all forms of illnesses, medical and surgical, acute and chronic, it is the possibility of the failure of the circulation which causes the doctor methodically to examine the pulse.

Before we can give a prognosis, or undertake an inquiry into prognosis, we must have a clear conception of heart failure, the manner of its production, and the signs by which it can be recognized.

The manner of death.—But first it is necessary to understand how death is brought about from heart failure, since it is the knowledge that heart failure

leads to death which is at the back of the minds of both doctor and patient.

Death is only considered to take place when the heart ceases to beat. The reason for this is that the vitality of all those organs which maintain life is dependent on an efficient supply of blood. As the heart is the organ which does this service, its integrity is ever a matter of essential moment.

In order, therefore, to be able to assess the value of symptoms in relation to the danger to life, it is necessary to recognize the organs on whose functional integrity life depends, or on whose failure death results. As death occurs when the heart ceases to beat, this cessation is brought about in three ways :

(1) From conditions within the heart itself.

(2) From failure of the respiration.

(3) From failure of the circulation on account of dilatation of the capillaries.

The cessation of the heart-beat. (*a*) *From conditions within the heart.*—The heart may cease to beat in a number of ways. The best conception of how this comes about is found in the death of those people whose powers gradually weaken in consequence of old age or wasting diseases. With advancing years there is a gradual but universal obliteration of the capillaries associated often with degenerative changes of the arteries, probably also due to the obliteration of the capillaries supplying the arterial wall. In consequence of these changes the blood supply to the organs becomes diminished, so that the capacity of the different organs to meet the calls of life becomes gradually but surely restricted. This limitation of the blood supply proceeds at a greater rate in some organs than in others. As the blood supply to the heart muscle diminishes, the functional capacity of the ventricles gradually decreases.

As the changes which limit the blood supply are steadily progressive, it is only a matter of time until the contractions of the ventricle become so feeble that the blood supply is not sufficient to nourish those organs which are essential to life.

This enfeeblement of the heart may occur earlier in life, and proceed at a greater rate when the heart muscle is the seat of active disease, or when there are, in addition, impediments to its work, as when the valves are diseased. Other conditions may hamper the damaged heart muscle in its work, as the disturbances in regulation and co-ordination of the different parts concerned in the heart's contraction. The heart muscle may become weakened by toxins of disease and by impaired nutrition. The heart ceases to beat from such causes by gradual exhaustion of the heart muscle.

The stoppage of the heart may take place on account of the cessation of impulses to the ventricle, as in heart block. Here the ventricle stands still, and the output of blood is stopped and death results. The ventricle may pass into a state of fibrillation, and as the ventricle is then ineffective the output of blood ceases, and death speedily follows.

It will thus be seen that heart failure and death are here due to the functional incapacity or inactivity of the *heart muscle.*

(*b*) *From the respiration.*—While such is the manner in which death is

brought about, there are two other systems in the body the suspension of whose activity leads directly to death. The chief of these is the suspension of the respiration. So long as the heart is supplied with suitable nourishment, of which oxygen is an essential constituent, the heart will continue to beat, even if taken out of the body, but if it does not receive oxygen, as when the breathing is suspended, the heart speedily becomes embarrassed and ceases to beat. Any condition which imperils the activity of the respiratory system leads to the cessation of the heart-beat and death.

(*c*) *From the capillaries.*—Another method by which the heart can cease to beat comes through the cessation of activity of the capillary system. The capacity of the capillaries when they are fully dilated is so greatly increased that they easily contain all the blood of the body. When this happens, as it sometimes does in the course of malignant infections or 'shock', there is no blood for the heart to put out, the pulse becomes feebler and feebler, and the heart gradually ceases to beat.

CHAPTER V

On measuring the Functional Efficiency of the Heart

There are fields in medicine a knowledge of which is essential to rational practice, but which are either ignored or so little understood as not to be available. One of these is the functional impairment of organs.

Ill-health is really due to the functional impairment of organs, which, in turn, may be the consequence of structural changes in one or more organs, or of disturbance of function, so that the organ or organs do not play their part in that harmonious activity which is necessary for health.

It is true that the need for a knowledge of the functional efficiency of organs has already been recognized, and endeavours have been made to measure the functional efficiency of certain organs, as the stomach, kidneys and heart. These attempts have, in the main, failed. No doubt, these endeavours have thrown some light on certain features of the impaired function, but on the whole little useful knowledge, and much that is useless and even misleading, has been acquired.

When we inquire into the cause of the failure, it will be found to be due to our ignorance of the structures and processes by which function is regulated, so that such tests as might reveal the function of the organ cannot be employed. We all know how little help test meals are in bringing to light the functional efficiency of the stomach. They may reveal some abnormality, but they are often valueless and apt to be misleading. The reason for failure is obvious. The test meal should be one that calls for the varied functions which the organ employs to accomplish its normal work. A test consisting of some unstimulating material, as milk and egg or a solution of oatmeal, does not call for the various secretions necessary to digestion. We find the stomach of a healthy and vigorous man may refuse to secrete hydrochloric acid in response to such a meal, with the result that he may be said to have achylia! But place before him a plate with steak and onions, and the juices will flow abundantly.

In the same way the reaction of the kidney to various tests, as urea and other chemicals, may make a call on but a limited number of its functions. The employment of such substances can only give scant information as to the power of the kidney to deal with its routine daily work. But even though such tests may be found at times of value, they do not carry medicine far, in that they throw no light upon the structures and processes which provide the evidences.

Numerous attempts have been made to find a standard for estimating the heart's efficiency. It has been supposed that the heart's efficiency can be tested by the individual performing some form of effort graded in some particular way, and an endeavour has been made to find signs to indicate the exhaustion of the heart's power. The effects of tests of various kinds, as hopping on one leg, lifting weights, etc., upon the rate of the heart, the rate of breathing, the blood-pressure, etc., have been employed to furnish this evidence.

Such tests and the conditions under which they are employed are just as incapable of bringing to light the functional efficiency of the heart as the tests used to find the functional efficiency of the stomach and kidney. When the enormous amount of work that has been expended to find the capacity of individuals for bodily work is considered and it is seen that none of the numerous methods have been found satisfactory, we are forced to recognize that medicine has not advanced far enough to know how such an undertaking should be approached.

Before we employ any test we must know the nature of our measure. The employment of a measure as a measure without knowing what it measures gives a useless kind of knowledge or leads to fallacious results. Because a foot-rule can measure a yard of cloth, it does not follow that it can measure a pint of beer. Because an increased rate of pulse may indicate the sensitivity of the sino-auricular node, it does not follow that it can throw light upon the functional efficiency of the heart.

I put the matter in this way to demonstrate that we must have a clear understanding of our measure. As we get a better insight into the mechanism of signs and symptoms it will be found that the variations in the rate of the heart and the blood-pressure have either no relation, or but an accidental relation to the heart's efficiency. Before we are in a position to determine the functional efficiency of the heart in any given case we must not only know what the standard measure is, but we must understand what heart failure is, what its symptoms are, and how it is brought about.

The failure of all those endeavours to find a test for the efficiency of an organ affords an excellent illustration of the statement that if an enterprise which requires a certain kind of knowledge be undertaken by people who do not possess that knowledge, failure is bound to result.

In all researches undertaken to find out the functional efficiency of an organ, the investigator must not only possess a knowledge of the factors concerned in the performance of its function ; he must be able to recognize the phenomena which result from failure of the organ. If this is not known, the likelihood is that the essential signs will be missed, while others will be used for purposes for which they are unsuited. Moreover, the tests to be employed should be those which correspond to the calls made upon the organ in the ordinary course of a normal life.

So far as the heart is concerned there is no hope of any one being able to estimate the condition of its functional capacity unless he understands

the nature of the phenomena which the heart exhibits, as well as the signs which result from heart failure.

In the description of the various symptoms evoked by the heart an attempt will be made to bring out the relation of the symptoms to heart failure. I know that this is a matter of great difficulty, because, had it been easy, it would have been done long ago. I do not assume that I have been successful in all instances, for this subject is only in a preliminary stage of development, and the principles which should guide to the recognition of essential matters are only slowly becoming recognizable. It is to be hoped that this aspect of the subject may appeal to some of my readers, so that they may be stimulated to carry the subject beyond the stage that I have reached. It may be taken for granted that until this kind of knowledge forms the subject of earnest research the progress of medicine will be seriously hampered.

CHAPTER VI

Heart Failure and the Response to Effort

The rest and reserve power of the heart.
The laws of ' all or nothing ' and fluctuation.
The response of the heart to effort.
The origin of symptoms of heart failure.
Symptoms of heart failure due to a deficient output of blood.
(*a*) Breathlessness.
(*b*) Orthopnœa.
(*c*) Cheyne-Stokes breathing.
(*d*) Cardiac asthma.
(*e*) Effects of diminished blood supply to the brain.
(*f*) Dropsy.
(*g*) Œdema of the lungs.
(*h*) Enlargement of the liver.
(*i*) Cyanosis.

In the last chapter it was shown that a knowledge of heart failure was necessary for the recognition of the functional capacity of the heart—in other words, of the ability of the heart to meet the requirements of the body in the performance of its daily routine. The special structure concerned in supplying blood to the organs of the body is the heart muscle. When it does not do this efficiently the heart is said to fail. The signs of heart failure are shown by the organs whose blood supply is insufficient and *are not to be found by the examination of the heart.*

The signs which are usually taken as distinct evidences of heart failure and which are sometimes spoken of as the ' cardinal ' symptoms of heart failure are dropsy, enlargement of the liver, orthopnœa and cyanosis. These, however, are the symptoms of advanced heart failure, and usually long before they are shown there are other evidences quite distinctive. Indeed, most people who suffer and die from heart failure never show these signs.

The rest and reserve power of the heart.—In the early days of my investigation into the efficiency of the heart, I divided the heart's power into two classes. One, which I called the ' rest power ', sufficed to maintain an efficient circulation when the body was at rest; a second, a ' reserve power ', enabled the heart to maintain an efficient circulation when bodily effort was made. Heart failure sets in by a gradual limitation of the reserve power. This is shown by the individual suffering distress from an effort which hitherto he had been able to undertake with comfort.

For a long time I was not able clearly to comprehend on what basis this true and useful method of estimating the heart's power was laid. In the course of a later inquiry the reason has become clear.

The laws of 'all or nothing' and fluctuation.—The difficulty in being able to understand the meaning of the rest and of the reserve power was, in a measure, due to the conception of the law of 'all or nothing'. Holding this view that when the heart contracted all the muscle cells participated, one could not understand how it came about that a heart was able to vary the amount of work it performed. It has been found, however, that this law does not apply to the normally acting heart; it is an experimental result. Only a portion of the muscle cells participates in each contraction of the ventricle. When the body is at rest a relatively small proportion participates in the contraction; when effort is made a much larger proportion takes part in the contraction. That is to say, the normal heart in its contraction is guided by the law of 'fluctuation' (see page 64), and not by the law of 'all or nothing'.

With the recognition of this law of fluctuation we obtain a clear conception of heart failure and its relation to the response to effort. The recognition of this law leads to the further inquiry into the manner in which the ventricular rate and contraction are adjusted to the requirements of the body.

The response of the heart to effort.—The various structures which go to make up the heart are specially adapted to favour an increase or decrease in the strength of the heart's contraction. The blood-vessels of the heart dilate to supply a greater amount of blood to the muscle fibres. The genetic system is reflexly stimulated by impulses from muscles of the body wall, which increase the rate and at the same time increase the number of muscle fibres that take part in the contraction.

How the limitation of the response to effort is produced.—Any condition which diminishes the number of muscle cells held in reserve when the body is at rest favours the onset of heart failure by limiting the power of the heart to respond to effort. Such limitations may come about in different ways, such as by the destruction of muscle cells from the various forms of disease, or by a diminished blood supply, through disease of the blood-vessels, or the presence of obstacles to the heart's work, as diseased valves.

Early production of heart failure.—An efficient circulation may be maintained with a damaged heart muscle when the body is at rest, but when effort is made the circulation is no longer efficient. The early stages of such inefficiency will be shown only when the heart is called upon to make an effort beyond its capacity, that is when a call is made for more muscle cells to work. This will be found to occur when an effort is made which the individual was wont to accomplish without distress. As impairment of the heart proceeds the individual's power of response becomes gradually more and more restricted, until the slightest effort produces distress and life itself becomes imperilled.

The origin of the symptoms of heart failure.—This explanation of heart failure is not the result of speculation, but is the outcome of a long-continued

investigation. Such an investigation requires appropriate methods. The method for this investigation required making careful records during life and correlating them with the conditions found after death. This may be said to be the usual procedure in clinical medicine, but such a method has to be carried out with some intelligence. It must be known what to record. The method of making a note of every sign or symptom which strikes the observer without any guiding principle results in a mass of phenomena being recorded, none of which may have any relation to the object of investigation, while, on the other hand, the essential phenomena may be, and often are, overlooked.

This aspect of the matter was forced upon my attention early on in my investigations into heart failure. A patient whom I had been observing for years died suddenly. At a post-mortem examination there was found a cavity in the wall of the left ventricle due to a disappearance of a large portion of the muscle, evidently the result of the blocking of a large branch of the coronary artery. When I reflected upon this condition it was evident that it must have been present for a long time and must have impaired the functional capacity of the heart, and the question arose, what were the evidences produced by this impairment? I had taken a large mass of records, but there was nothing distinctive to be made out in the notes of the physical examination of the patient. The heart was not enlarged, the rate and rhythm were perfectly normal, both when at rest and on making an effort, and nothing distinctive could be found in the sounds of the heart. His response to effort in the later months of his life had been greatly limited because of pain. This, with some associated phenomena such as a profuse flow of saliva when the pain was present, was the only indication of the heart's impairment. Indeed it was the only sign by which the functional capacity of the heart could be estimated.

At the same time I had a series of cases which terminated in death, and the post-mortem examination revealed extensive fibrotic degeneration of the heart's muscle; and again looking up my notes to see what were the evidences of such changes during life, I found in some the only physical change was a slight increase in the size of the heart, while the patient's response to effort was limited by breathlessness, and as death approached, dyspnœa was a distressing symptom.

In another set of patients attacks of breathlessness came on, and at the same time the heart was found to be beating at a rapid rate with a very irregular rhythm, with many small imperfect pulse beats. As soon as the rate became normal and the rhythm regular the breathlessness at once disappeared, so that there could be no doubt that the distress was connected with the abnormal pulse beats. These were so small that it was manifest that the output of blood was defective.

In these instances one could safely infer that the output of blood was not sufficient to supply the organs of the body with enough blood to maintain their functions unimpaired, consequently the symptoms of heart failure were not exhibited by the heart, but by the organs whose blood supply was deficient.

Proceeding upon these lines the view was gradually evolved, first, that

the symptoms of heart failure are not to be found by an examination of the heart; second, that the early symptoms are only to be found when the heart is compelled to respond to effort, and that they are not exhibited by any physical sign, but by the patient's sensation of distress; third, these sensations of distress are of two kinds—those provoked by the heart muscle, which result in pain, and those provoked by a defective output of the heart, which result in symptoms produced by the organs which suffer from the defective output, the chief of these being the symptoms produced by the respiratory system.

The conclusion arrived at from these considerations is that heart failure is shown by a limitation of the field of response to effort, and what limits effort is the sensations of the patient. This conclusion will serve as a guide in the description of affections of the heart in this book. The essential matter of heart failure will always be kept in view—the various phenomena will be described so as to bring out their relation to the production of heart failure.

As the symptoms of heart failure are in the main sensations of distress experienced by the patient, of which pain and breathlessness are most distinctive, the question of pain in relation to heart failure will be discussed at some length in the chapter on Angina Pectoris. The symptoms provoked by a failure of output will be more briefly dealt with here, and referred to in the description of symptoms of individual patients. I could have wished to discuss the various forms of breathlessness at some length, but although I have tried to get a better understanding of them, I have not been successful, and therefore merely refer to them as being undoubtedly produced by heart failure.

The symptoms of heart failure due to a deficient output of blood.—(*a*) *Breathlessness.*—The symptoms of heart failure from deficient output of blood might be found in almost any organ did we possess the means of observing them. It so happens that one system which suffers early from an impaired blood supply is one which readily gives rise to distress. This is the respiratory system, and it is the distress in breathing on response to effort which is usually the earliest sign of heart failure. As the heart failure proceeds the distress in breathing becomes more easily provoked, until a stage is reached when it is present even when the body is at rest.

(*b*) *Orthopnœa.*—A distinctive sign of extreme heart failure is orthopnœa, the failure having gone so far that the patient suffers such distress in breathing on lying down that he is compelled to sit, or be propped up.

(*c*) *Cheyne-Stokes breathing.*—Another form of distressed breathing is Cheyne-Stokes respiration. Many elderly people exhibit Cheyne-Stokes respiration without being conscious of it. But in certain forms of heart failure it may occur and become so marked that the patient's rest is disturbed during the period of increased breathing. This is preceded by a period when the breathing almost ceases, or completely ceases, it may be for a period of half a minute to a minute, during which the patient may fall asleep, to be wakened up, it may be, by a sense of suffocation; on its resumption the breathing is often exaggerated.

(*d*) *Cardiac asthma.*—Occasionally the periods of apnœa are so complete and prolonged that the patient wakes up with a sense of great distress, sometimes so severe that he may spring out of bed in his extremity, and may then have to breathe heavily and in laboured fashion for many minutes. This is the condition which has received the term ' cardiac asthma '.

(*e*) *Cerebral symptoms.*—While the foregoing description has reference to the more common forms of symptoms produced by a defective output of blood, there are occasionally symptoms produced by other organs, chiefly the brain, which give rise to symptoms such as giddiness and loss of consciousness. These will be dealt with later.

(*f*) *Dropsy.*—The causes of dropsy are still obscure. The fact that it is found in heart failure in dependent parts of the body indicates that certain forms of it are due to a diminution of the force which propels the blood through the capillaries, and there is no doubt that it is of frequent occurrence where the heart's force is greatly enfeebled, as in auricular fibrillation with numerous small and ineffective beats. There are, however, conditions of the same kind without any dropsy. It is also not uncommon with high blood-pressure; but here it is possible that the contraction of the arterioles, which is a factor in raising the blood-pressure, may diminish the force in the capillaries.

In cardiac dropsy œdema begins about the ankles and in the legs when the patient is out of bed. When lying in bed it is found across the sacrum. Dropsy may increase till the legs and external genitals are greatly swollen.

Ascites and hydrothorax may sometimes occur without much œdema elsewhere. There is seldom œdema of the arms and face. If it is found in one arm it may be because the patient lies on that side. When not due to this cause it will be found to be due to clotting in the jugular and neighbouring veins.

(*g*) *Œdema of the lungs.*—When patients with enfeebled hearts are kept in bed the circulation in the lungs becomes embarrassed, the pressure of the ribs on the bed preventing the free movement of the lungs, so that the enfeebled right ventricle cannot force the blood through the immobile part of the lung, and œdema sets in. It is shown first by fine crepitations heard on auscultating the bases of the lung behind. In the early stages they disappear after a few deep breaths, but with progressive failure of the heart the œdema extends, the bases of the lung become dull, and the patient may expectorate blood-stained sputum, and at the post-mortem examination the bases of the lung are found sodden and airless.

The conditions in which œdema of the lungs is most commonly found are in mitral stenosis, in patients suffering from an exhausting complaint like typhoid fever, in elderly people kept in bed for some reason, as a broken leg or a surgical operation, and in attacks of abnormal rhythms such as auricular flutter and fibrillation.

(*h*) *Enlargement of the liver.*—The blood circulating in the liver which has come through the portal system passes through two sets of capillaries, so that the force exerted by the heart in the second set of capillaries within the liver,

is so feeble that the blood tends to stagnate, and the liver enlarges. The capacity of the liver to enlarge is very remarkable. After the onset of an attack of heart failure, such as occurs in auricular fibrillation and flutter, the liver may in a short time be found to extend below the level of the umbilicus. The restoration of the normal rhythm, or the slowing of the rate under the influence of digitalis, causes the enlargement speedily to disappear.

This remarkable tendency to enlargement of the liver is evidently related to its peculiar function in seals and other diving animals, where there is a special provision for the blood to be stored in that organ when the animal is under water.

In the early stages of enlarged liver from heart failure there may be great tenderness of the skin and deeper structures of the external body wall over the region of the liver.

(*i*) *Cyanosis.*—Cyanosis is another symptom which it is difficult to explain. In certain forms of heart failure, particularly in that due to auricular fibrillation, it is often very marked, the nose, lips, and particularly the lobes of the ears, becoming dark blue. Here it is manifestly due to the driving force being just able to send the blood through the capillaries, but so feebly that the blood stagnates in the venules. But there must be some other agent at work, for patients with auricular fibrillation may die from heart failure with little or no cyanosis.

CHAPTER VII

The Principles of Investigation

The problem of ill-health.
The end result.
The need for a new concept.
The factors concerned in vital activities.

The problem of ill-health.—Before entering upon his enterprise an investigator should clearly define his aim and object. Having done so, he should next consider whether he possesses the knowledge requisite for its accomplishment.

The ultimate aim of all medical investigation is to combat ill-health. Health is dependent upon the efficient functioning of the organs of the body, while ill-health is due to their impaired functioning.

If a would-be investigator into any of the problems of ill-health desires to qualify himself for this enterprise, he must first learn the manner in which organs perform their functions. He may imagine that this knowledge has already been acquired, but as he proceeds in his investigations he will discover that this knowledge is too defective for his purpose.

This may seem a somewhat exaggerated statement in view of the great amount of research into the functions of the various organs, in nearly every department of medicine. Nevertheless, although a great many facts have been brought to light, this knowledge is so defective that at present it is not possible to understand how organs normally perform their functions, whether it is a simple organ like a capillary or a complex organ like the heart or the kidney. As a consequence it is practically impossible to tell the nature of any reaction, whether it is the manifestation of a healthy organ, or a symptom of disease with that particularity which is necessary for understanding the nature of ill-health.

The end result.—The reason for this defective knowledge can be traced to the custom in every branch of medicine, of limiting inquiry to the production and recognition of end results. Such end results may be a reaction to an experiment, the effect of a drug, or a symptom of disease, but practically in no instance is there to be found a description of how such an end result is brought about.

An end result is the outcome of a series of events involving the participation of a number of structures and processes. For the proper understanding, therefore, of an end result it is necessary to know in each case what these structures and processes are. A slowing of the heart-rate may take place as a

normal event, as the result of an experiment, as the effect of a drug or of disease, but to-day it is not possible to tell what are the structures and processes involved in the manifestation.

The need for a new concept.—The lack of this kind of knowledge at a time when an enormous amount of attention is being given to every phase of medicine, indicates that the conception of research which is dominant to-day is inadequate, and that some other concept must be introduced to make good such defect.

It is one thing to recognize the need for such a concept, but it is another thing to find it. The need has been apparent to me for many years, and it was one of the first problems we attacked when we started the Research Institute in St. Andrews. We realized that to pursue the methods which had become stereotyped, of searching for new phenomena, and devising new instruments for that purpose, would be futile, for the number of phenomena produced by disease already recognized is so great, and the mass so chaotic, that the mere addition to them would but add confusion to a subject already confused.

The factors concerned in vital activities.—To obtain a concept which will enable an investigator to acquire a knowledge of the manner in which the organs function, it will be well to realize what are the two fundamental factors concerned in the functioning of organs, namely, living structures and vital processes.

While every one will recognize that these two factors are essential to vital activities, hitherto it has not been possible to investigate them in such a way as to reveal the part they play.

In the description of the 'end result' it was stated that in its production a number of structures and processes participated. As a guide to an investigation for the purpose of discovering these structures and processes, we have adopted a method which we have called 'the application of the principle of the reflex arc'. In the following chapter this principle is described and its application illustrated in the interpretation of certain irregularities in the heart's action.

While this method reveals the structures and processes, these latter are so imperfectly recognized that it has been found necessary to undertake an investigation into their nature. The method of investigation and the results obtained will be described in Chapters IX.–XII.

Following upon the investigations on these lines, the method of production of symptoms is sufficiently recognized to enable a classification to be made of the chaotic mass of symptoms according to their affinity in nature—the only kind of classification which is of real scientific value. This is described in Chapter XIII.

CHAPTER VIII

The Reflex Arc

The constitution of the reflex arc.
The meaning of the reflex arc.
The application of the principle of the reflex arc.
The muscular and genetic systems of the heart.
The part played by the genetic system.
The application of the reflex arc.
The application of the reflex arc to the interpretation of heart irregularities.
The value of the interpretation of symptoms by means of the reflex arc.

The constitution of the reflex arc.—The constitution of the reflex arc can be briefly recapitulated. Cells only discharge their function in response to impulses from without. Every organ of the body possesses certain structures on which its functional activity depends. There is, first, the structure, consisting of cells which give rise to the peculiar function of the organ, such as contraction, secretion, sensation. In the reflex arc this is called the ' effector ' organ.

There are, second, structures which originate and convey the impulses which stimulate the functional cells into activity, and at the same time regulate this activity according to the requirements of the body.

It is upon these structures or upon the cells of the effector organ, but mainly on the former, that the agents of disease act when they produce ill-health and death. It is also upon these structures that all kinds of remedies act.

When, then, we set out to find where the disturbance arises which produces an end result, a knowledge of the structures concerned in producing the stimulus and conveying it to the effector organ is necessary. The application of the principle of the reflex arc has revealed that the normal regulation of functional activity is so imperfectly understood that we have no knowledge of this kind in regard to a single organ of the body which is sufficient to solve the simplest problems in the practice of medicine.

The meaning of the reflex arc.—All vital processes are carried on by the stimulation of a living cell by an impulse. The impulse may reach the cell in a variety of ways. It may be communicated from a neighbouring cell without the intervention of any other structure. It may come from a distance by a series of structures specially evolved for the purpose, such as the nerves. This process of impulse conduction and reaction is commonly spoken of as a ' reflex ', and all these structures which lie between the source of the impulse and the organ on which it acts are elements of the reflex arc. This may be represented

by a simple diagram, as in Fig. 1. In this diagram *A* is the structure whose activity discharges impulses. At *B* are the fibrils (receptors) which receive these impulses. At *C* are the fibrils which discharge the impulses to the organ *D*, which responds by discharging its function (effector organ).

The structure which connects the disturbed organ (*A*) with the effector organ (*D*) is usually a nerve structure, but in the heart there is a mechanism which effects the same purpose in a different manner, and whose structure is different from that of a nerve. This has been called the 'conducting system', but, as the term is inappropriate and even misleading, it is proposed, for reasons to be given later, to call it the 'genetic system'.

This description of the reflex arc has become stereotyped, and save for convenience in explaining reactions it has been of no service. But we have employed it for other purposes, and found it of the utmost value as a method and guide to the solution of medical problems. Indeed, the 'application of the principle of the reflex arc' provides practically the only procedure which enables medical investigation to be carried on in a rational and systematic manner.

The application of the principle of the reflex arc.—By the application of the

FIG. 1. Diagram of a simple reflex arc. *A*, the structure in which the disturbance arises and which discharges the impulses; *B*, the fibrils which receive the impulses; *C*, fibrils which discharge the impulses to *D*, the effector organ.

principle of the reflex arc is meant the analysis of the arc to find out the structure in which the disturbance arose and the nature of the vital process which resulted in the production of the symptom.

This proposition seems so simple, yet it has been strangely overlooked, for a great many investigations have been devoted to looking for a cause of a reaction or a symptom in the structure which exhibited it, in place of searching for the source of disturbance of which the reaction or symptom was but an expression.

Each element or structure of which the arc is composed is made up of living active cells, and is liable to the variations which occur in every organic structure. Consequently, the disturbances which produce the symptom may arise in any one of the structures concerned in the reflex arc, in the organ which produces the impulse or the structures which receive the impulses, or in any of the various structures which convey the impulses to the effector organ which exhibits the symptom.

The muscular and genetic systems of the heart.—Before going further it may be well to illustrate what is meant by the application of this principle, although it anticipates matters which will be dealt with later in some detail. The heart affords an excellent example for illustration, as the parts which are concerned in its movement are relatively easy to recognize.

These parts are the muscle which exhibits the symptoms, and the structures which regulate and control the contraction of the muscles.

These regulating structures are recognized as the sino-auricular node and the auriculo-ventricular node and bundle (Fig. 2). It is customary to speak of the impulse being conveyed from the s.-a. node by these structures to the

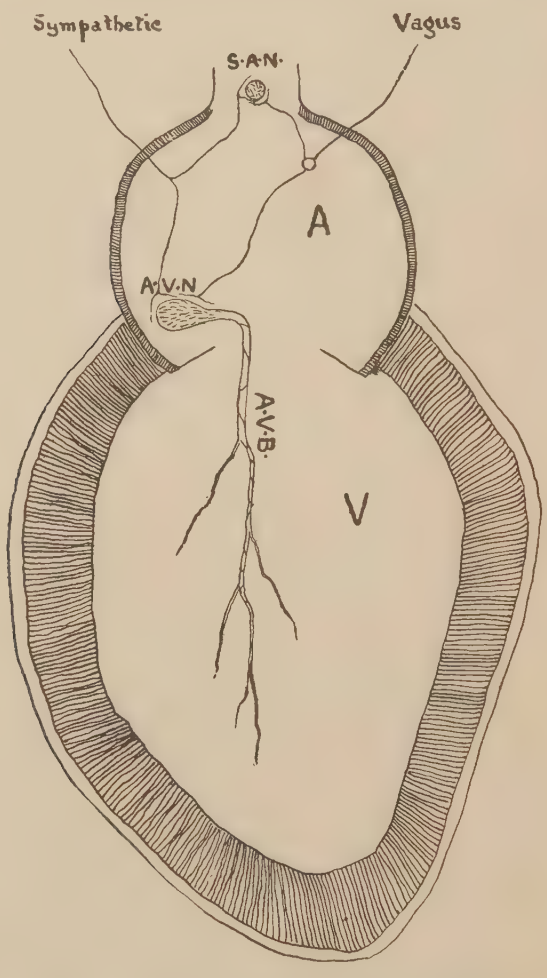


Fig. 2. Diagram of the structures concerned in the movements of the heart. *V* is the ventricle and *A* the auricle. *S.A.N.* is the sino-auricular node, *A.V.N.* the auriculo-ventricular node, and *A.V.B.* the auriculo-ventricular bundle. The sympathetic and vagus are represented as being distributed to the two nodes, as it is on them they act in regulating the rate and rhythm of the heart. (From the *Reports of the St. Andrews Institute*, vol. ii. p. 10.)

ventricle, and to describe them as parts of the 'conducting system'. But this term is misleading, for the cells do not convey impulses after the manner of a nerve. They pass the impulse along by first contracting, then discharging an impulse so that one cell stimulates another until the impulses reach the

muscle fibres (see p. 61). In addition, this system has other functions which will be elucidated later,

The part played by the genetic system.—The genetic system is concerned in originating impulses and in conveying them to the different chambers of the heart, and in regulating the contraction of these chambers, in a peculiar manner. Seeing that no organ exercises its function unless stimulated by impulses conveyed to it by other structures which have been specially evolved for the purpose, all manifestations of functional activity are evidences of the activity, not only of the structures showing the manifestation, but also of the structures that originated the impulses and conveyed them to the organ.

This is seemingly so evident as scarcely to need comment, but it is a very important conception for the interpretation of symptoms, inasmuch as it brings home to us the fact that we must look for the cause of the symptom beyond the structures that exhibit it.

A knowledge of the functions of the genetic system of the heart is only to be attained by recognizing and interpreting the movement of the auricles and

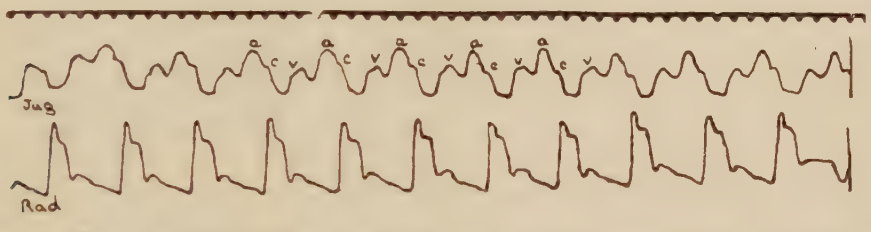


FIG. 3. Tracings of the radial and jugular pulses with the normal rhythm. In the jugular tracing the waves *a* are due to the auricle and the waves *c* to the carotid. (The time marks in this, as in all other tracings, are $\frac{1}{5}$ of a second.)

the ventricles or their lack of movement. The auricle is under the control of the sino-auricular node, so that we can tell what this node is doing by observing the behaviour of the auricle. The ventricle is normally under the control of the s.-a. node, through the auriculo-ventricular node, so the behaviour of the ventricle informs us of what one or other of these nodes is doing.

The application of the principle of the reflex arc.—The structures concerned directly in the heart-beat being the genetic system and the auricles and ventricles, they can be represented in a diagram where the relation of the one to the other can be illustrated. A tracing of the jugular and radial pulses [1] (Fig. 3) reveals the movements of the right auricle and left ventricle. As the auricles act together and the ventricles together, the action of the one, auricle or ventricle, tells us what the other is doing. We can therefore indicate the movements of the auricles and ventricles as shown in Fig. 3 in a diagram (Fig. 3A), where the downstrokes in the space *As* represent the auricular contraction and the downstrokes in the space *Vs* represent the ventricular contraction.

As the movement of the auricle is dependent upon the s.-a. node, this node

[1] Descriptions of the manner of taking and interpreting graphic records are given in Chapter LXIV., p. 418.

can be represented in the space *S.A.N.*, and as the ventricle is dependent upon the a.-v. node, that structure can be represented in the space *A.V.N.*

In the space *S.A.N.* there is a sharp line followed by a shaded area, dark at first, and gradually becoming lighter—the downstroke represents the dis-

Fig. 3a. A diagram of Fig. 3, showing the part each element of the conducting system plays in the normal heart-beat. In this diagram, as in all the other diagrams, the upper space (*S.A.N.*) represents the sino-auricular node. The shading represents the refractory period after the discharge of the impulse. The shading gradually fades, indicating an increasing susceptibility of the node to stimulation. The discharge of the node causes the contraction of the auricle (waves *a* in the jugular tracing) represented by the downstrokes in the space *As*. The impulse passes to the auriculo-ventricular node (space *A.V.N.*), which passes it on to the ventricle and causes the ventricle to contract. The contraction is represented in the space *Vs* (corresponding to the *c* wave in the jugular and the radial pulse).

charge of the impulses to the auricle, while the shaded area represents the period of exhaustion, and the gradual recovery, represented by the fading of the shading, till the recovery is complete and other impulses are discharged.

In response to the discharge of an impulse there is an auricular contraction represented by the downstroke in the space *As*.

Following the auricular contraction the impulses reach the a.-v. node, which is stimulated into activity and in its turn discharges impulses which are conveyed to the ventricle and result in its contraction. This contraction is represented by the downstrokes in the space *Vs*. From the presence of the ventricular contraction we reason that the a.-v. node has discharged its impulses. The a.-v. node is so dependent upon the s.-a. node in a normal acting heart that we have no evidence of its exhaustion and recovery.

The application of the reflex arc to the interpretation of heart irregularities. —The various forms of irregularities when represented in this diagrammatic fashion reveal the source of the disturbance.

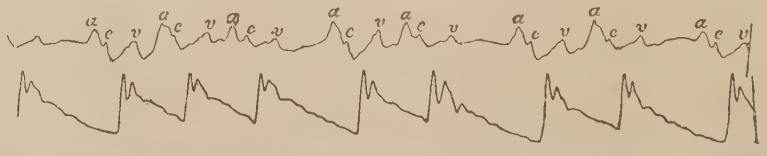


Fig. 4. Simultaneous records of the jugular and radial pulses, showing an irregularity in the rhythm of the pulse in which all the chambers of the heart participate. This tracing is interpreted in the diagram Fig. 4a.

In Fig. 4 there is shown in the radial pulse an irregularity. In the upper tracing the waves *c* are due to the carotid and they are seen to correspond with the radial pulse, and the waves *a* are due to the auricle and immediately precede the carotid pulse. These events can be represented in the form of

a diagram in Fig. 4A. If we trace back the incidents from the ventricle, we find that the a.-v. node participates in the irregularity. The auricle shows the same irregularity, and as it is dependent on the s.-a. node, we thus trace the disturbance to the s.-a. node, which is represented as recovering from the

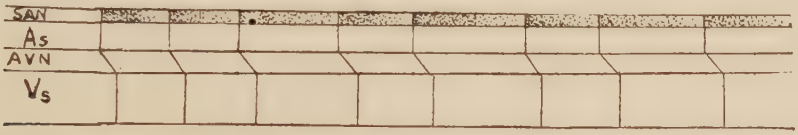


FIG. 4A. A diagram of Fig. 4, showing that the irregularity is due to the variations in the time of recovery of the sino-auricular node.

exhaustion at varying periods, as shown by the shading. In this way we can recognize that some agent was modifying the rate of recovery of the s.-a. node, and this leads to the inquiry of what agents are capable of inducing this variation. This brings into consideration the influence of the sympathetic and vagus nerve, which have a marked influence in modifying the rhythm and rate of the heart.

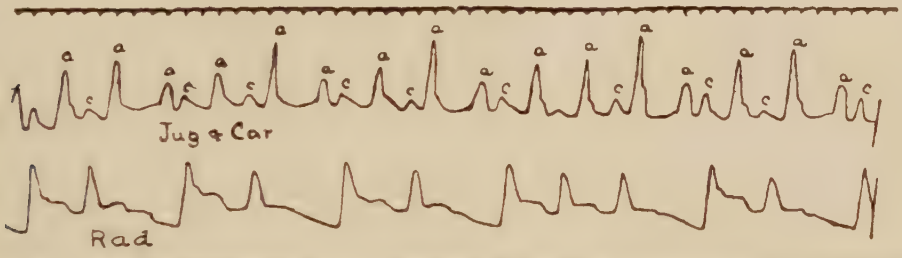


FIG. 5. A simultaneous tracing of the pulsation in the neck and of the radial in partial heart-block. The radial pulse (Rad) and carotid pulse (*c*) are irregular, but the auricular waves (*a*) are quite regular in time; the reason of this is shown in Fig. 5A. The waves *a* will be seen to vary in height, and this is due to the fact that the auricle contracts at times during the diastole of the ventricle and produces the small waves, and during the systole of the ventricle, when they increase in height.

FIG. 5A. Diagram of tracing in Fig. 5, showing where the disturbance took place which caused the irregular pulse. The sino-auricular node causes an auricular beat at regular intervals (space *As*). The auriculo-ventricular node (space *A. V.N.*) is seen to vary in its periods of recovery, so that occasionally an auricular beat falls out, when the node is not susceptible. In consequence the ventricular systole drops out and the pulse becomes irregular.

An extension of the diagram would embrace their activity and lead to the source of disturbance, but the demonstration here is limited to the genetic system.

In Fig. 5 the radial pulse shows an irregularity having a somewhat superficial resemblance to that of Fig. 4. If we construct a diagram (Fig. 5A)

representing the events shown in the jugular tracing, we discover that the ventricle is irregular and the a.-v. node is acting in a similarly irregular fashion, but we find the auricle as represented by the downstrokes in the space *As* is quite regular, so that we have no hesitation in recognizing the source of this disturbance to lie in the a.-v. node, and to be due to variations in the rate of its recovery.

In Fig. 6 there is shown another pulse tracing, extremely irregular, with a jugular pulse in which there is no auricular wave. Experience has shown

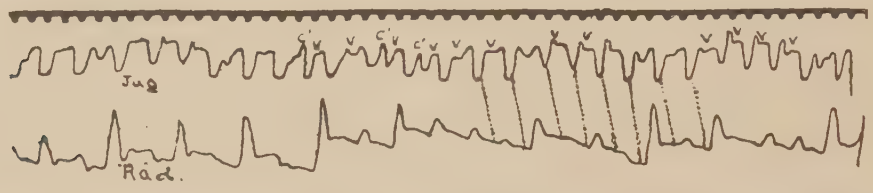


Fig. 6. Simultaneous records of the jugular and radial pulses. The radial pulse is irregular, of a kind typical of auricular fibrillation, inasmuch as there is no relation between the length of the preceding pause and the size of the beat. The waves (*v, c′ v*) in the jugular tracing are due to the ventricle, and are characteristic of auricular fibrillation in that they show no auricular wave. At times there is no evidence of a beat in the radial, but there is a *v* wave in the jugular, showing that the ventricles contracted, but the left so feebly that the wave in the radial was not sufficient to raise the lever of the sphygmograph. (See diagram, Fig. 6A.)

that this irregular pulse and this ventricular form of the jugular pulse indicate that the auricle is fibrillating. We can represent the ventricular systoles in the downstrokes in the space *V.S.* (Fig. 6A), and the a.-v. node is represented

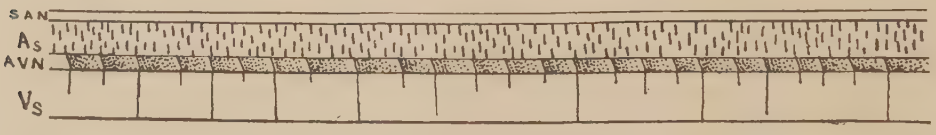


Fig. 6A. A diagram representing the events in Fig. 5. In the sino-auricular node space there is no sign of activity. The short strokes in the space *As* represent the impulses from the fibrillating auricle. The space *A.V.N.* represents the auriculo-ventricular node and shows its varying activity, and the irregularity of the pulse is seen to be due to the varying periods of recovery of this node. The downstrokes in the space *Vs* represent the ventricular systoles as shown in the radial pulse. These systoles vary in size and in force. The length of the strokes represents the output of the heart as shown in the radial tracing.

as originating these contractions. In the space *As* are a number of short strokes representing the fibrillating auricle, from which there is a shower of impulses. If we want to represent the s.-a. node, we discover that there is no evidence of its presence, and therefore the space *S.A.N.* is represented as a blank.

The application of this principle to the interpretation of the varied phenomena to be found in auricular flutter gives us a further glimpse into vital processes. In this condition the auricles beat at a rate about 300 per

minute. The radial pulse in Fig. 7 is typical of auricular flutter. The jugular pulse showed waves at the rate of 300 per minute. Some of these are seen in Fig. 9. They are represented in the space *As* in the diagram (Fig. 7). The radial pulse beats are at the rate of 150 per minute, and they are represented

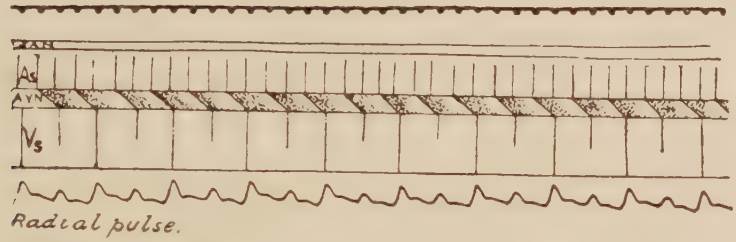


FIG. 7. Tracing of the radial pulse during auricular flutter. The diagram shows the parts concerned in producing this type of pulse. The sino-auricular node is out of action, as in Fig. 6A. The auricle is beating at twice the rate of the ventricle, as every second auricular impulse falls on the auriculo-ventricular node when it is refractory.

by the downstrokes in the space *Vs*. It will be seen that the radial pulse is alternating, and this is represented in the diagram by a long downstroke alternating with a short downstroke.

The reason for the rate of the ventricle being exactly half of the auricle is shown in the space *A.V.N.*, the shaded portion in that space representing the refractory period, and it is seen that every second beat follows upon a shaded portion of the diagram.

This tracing was taken immediately after the patient had lain down. A few minutes after lying down the pulse became less frequent in rate and irregular. Fig. 8 is a tracing of the radial pulse, about five minutes after the

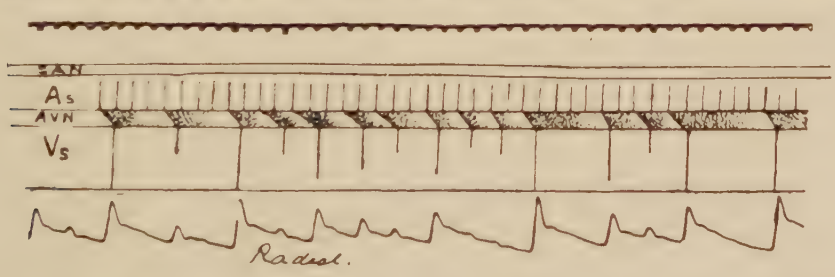


FIG. 8. Shows an irregular radial pulse—the auricular waves are constant, and the pulse irregularity is seen to be due to variations in the length of the refractory period of the auriculo-ventricular node. The radial tracing is a continuation of that in Fig. 7.

patient had lain down. It will be seen that it exhibits now a disorderly irregularity, as well as being slower. If we look at the space *As* we will find that the auricular waves are still constant, at a rate of 300 per minute, but if we look at the space *A.V.N.* we will find the shading varying in a very remarkable manner.

It is on account of the varying duration of the refractory period shown by the shading that the pulse is irregular, for a varying number of auricular beats fall during these shaded periods, and therefore fail to reach the ventricle. Five minutes later the record showed that the pulse had become still slower, falling to between 40 and 50 per minute, and that it was only slightly irregular (Fig. 9). We see that the cause of this slowing is due to an increase in the length of the refractory periods of the a.-v. node.

When the patient got out of bed the effort at once caused the pulse-rate to rise to 150 beats per minute, as in Fig. 7. These remarkable changes are diagnostic of auricular flutter, and will be commented on later (p. 217).

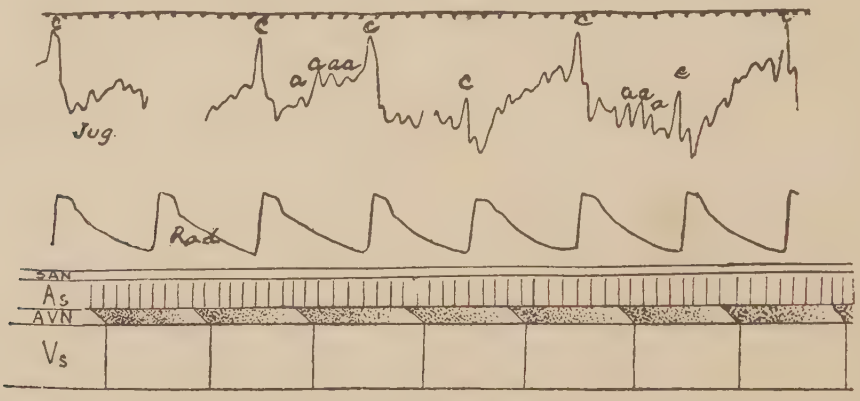


Fig. 9. The tracing is a continuation of Fig 8, and shows that the radial pulse has decreased still further in rate, due to a still greater increase of the refractory period of the auriculo-ventricular node. All these variations in the pulse-rate happened during a period of ten minutes, and the tracings are parts of that continuous record. When the patient exerted himself the pulse increased in rate at once to 150, as in Fig. 7. The jugular tracing shows some of the small auricular waves (*a*), which are represented in the diagram by the downstrokes in the space *As*. The *c* waves are due to the carotid.

The value of the interpretation of symptoms by means of the reflex arc.—The interpretation of these tracings by means of the application of the reflex arc shows much more than is described here. This way of looking at the matter opens up a new field for investigation, and throws a flood of light upon vital processes and the functions of organs. It affords the opportunity for a better understanding of such processes as stimulation, inhibition, excitability, the latent period, and the refractory state. It affords evidence that when the muscle cells contract they discharge impulses which can stimulate other cells to activity. They provide the key to the explanation of how effort and rest affect the heart-rate, a problem seemingly so simple, but hitherto insoluble. It affords the opportunity of studying the action of the vagus and sympathetic nerve. It shows the nature and origin of the imperfect and ineffective ventricular contractions which result in heart failure, the origin of certain irregularities, and it throws some light on the pulsus alternans. It gives evidence of the loss of control—an influence which is exerted by one structure

over another as that of the s.-a. node over the a.-v. node. It throws new light on the manner of the action of such drugs as digitalis. It enables a diagnosis to be made of auricular flutter without the aid of instruments, a condition hitherto one of the most difficult of all abnormal rhythms to diagnose.

The flood of light which has resulted from this method of investigation into such a limited field holds out the prospect that when applied to other fields it will guide research profitably in every department of medicine where at present research is limited to the recognition of end results.

It was the study of the irregularities in the way just given that led to the appreciation of vital processes described in the following chapter.

CHAPTER IX

The Vital Processes

Introduction.
Definition of the term 'vital process'.
The cell impulse and its investigation.
The basis for a classification of symptoms.

Introduction.—In the last chapter the structures concerned in producing an end result were discussed. It is necessary now to have a better understanding of what is meant by the other factor—the vital process.

The terms which indicate vital activity have hitherto been used as if they described the process, when as a matter of fact there was little understanding of the nature of any process. When we inquire into what is meant by such terms as stimulation, inhibition, contraction, conduction, tone, and so forth, it is found that they are merely descriptive of the superficial appearance of end results, or reactions. A reaction which showed increased activity was an evidence of stimulation of decreased activity, an evidence of inhibition. A shortening of a muscle was evidence of contraction. A reaction which appeared at a distance from a part stimulated was an evidence of conduction. A slight shortening or hardness of a muscle at rest was an evidence of tone. When it is desired to know how these various evidences of activity are brought about, there are offered only vague or misleading conjectures. To a great extent this imperfect knowledge of vital processes was brought about, as has already been stated, by investigators concentrating mainly on the contemplation of end results.

This defect of knowledge has been fostered to a great extent by the belief that the agent or force which was concerned in the production of these vital processes could be replaced by an artificial form of stimulation like electricity. It was not recognized that to employ a form of stimulation that Nature does not use is to get results that never occur in Nature. The consequence is that a great many reactions which are taken to be normal or physiological are, as a matter of fact, unnatural and artificial, and in this way the normal activities have been further obscured by interpretations in the light of abnormal reactions.

This does not imply that useful information has not been obtained from electrical stimulation. In addition to the reactions which satisfy the academic physiologist, it also supplies information useful to medicine. Evidence of

the organs supplied by nerves or the general function of such nerves as the sympathetic and vagus can be revealed by this method.

But while it gives evidence of this kind, the manner in which the reaction is brought about is different. The stimulation by electricity of the phrenic nerve gives rise to a contraction of the diaphragm, but the abrupt contraction of the whole muscle which results is quite different from the peculiar graded character of the normal contraction.

That there should be this difference can readily be recognized if we reflect that the impulse which Nature employs is different from electricity—as will be shown later—while the impulse enters the nerve by a part of the nerve specially evolved for the purpose and which cannot be replaced by stimulation applied to the stump of a cut nerve.

Definition of the term 'vital process'.—This term is used in the following sense. By a 'process' is meant a method of action. The combination of an acid and an alkali is spoken of as a 'chemical process'. Certain chemical processes can be hastened or delayed by heat and cold. The discharge of energy and its renewal by a living cell are due to chemical changes, and the process involved can be hastened or delayed by heat and cold. But there is another agent which acts in the same way which is only produced by living cells—the cell impulse. This impulse exercises an influence which not only hastens or delays the process, but regulates or controls it in a particular manner. As this form of activity is due to living cells, thus it is spoken of as a 'vital' process. 'Stimulation' indicates that the process is hastened, and 'inhibition' that it is delayed. When the activity is purposive, the process is said to be controlled. The factors concerned in the production of manifestations of vital activity are therefore living cells and vital processes.

The cell impulse and its investigation.—It has long been recognized that there is concerned in all forms of vital activity a force or agent which cannot itself be perceived, and its presence can only be inferred from the influence it exerts. In an investigation into this force, it was found to be generated by cells and to exert its influence on cells, and provisionally it is called the 'cell impulse'. It was also recognized that this agent must be like those forces in Nature which produce the varied phenomena exhibited by matter.

This led to the consideration of the line of investigation that should be pursued to discover the nature of this force. It was recognized that it would be futile to attempt to discover its nature by making it by itself an object of study. Much time and speculation had already been wasted on this endeavour. No one would seek to investigate the force of gravitation in this way, nevertheless the investigation of gravitation and other forces in Nature has led to a great addition to the knowledge of matter. This knowledge was acquired, not by the study of the force as a force, but by the study of the effects the force produced.

The basis for a classification of symptoms.—This was the method adopted in this investigation. We carefully noted all the evidences of vital activity, analysed them as far as we could, then when a sufficient number of observations

had been made they were carefully grouped and certain deductions drawn, which enabled us to recognize that certain results invariably occurred under particular circumstances. In this way we were gradually able to frame certain hypotheses which were provisionally adopted. We then proceeded to subject them to various tests, and there gradually emerged certain definite principles which enabled us to achieve one object of our research, namely, a recognition of the nature of symptoms which permitted classification of the symptoms of disease to be made according to their affinity in nature.

The most striking feature about this classification is its simplicity, but it is impossible to understand its significance or to appreciate its value until the nature of the vital processes is looked upon in a manner widely different from that in which it is regarded to-day.

The view of vital processes which is given here was obtained by the systematic investigation, at first of the more simple and easily observed structures, such as reaction of the skin to various forms of stimulation, the different ways in which voluntary muscles contract as seen by the clinical observer, and of the manner in which the capillaries carry on their work.

As our opportunities of studying the movements of the heart in health and in disease were abundant, we made use of these opportunities for studying the reaction of a more complex organ. In addition we used the opportunity of the study of all forms of ill-health.

In this way there was obtained a great mass of observations, and in their study there emerged, as has been stated, a few clearly recognized principles. The deductions derived from these observations were then applied to the study of manifestations of health and disease of other organs of the body, and to other animals and to the vegetable kingdom. It was found that these principles were applicable to every form of vital activity however varied the structure, and it is from this point of view that the following description of our results is given.

CHAPTER X

The Vital Activities in Plants

The method of investigating vital activities.
The principles on which the investigation is based.
The functions of plant cells.
- Stimulation.
- Contraction.
- Conduction.
- Inhibition.
- Control.

The cell impulse.

The method of investigating vital activities.—In describing a conception of vital activity it is advisable to begin with the less complicated structures, so that the source of any manifestation of activity can be referred to the particular element which produced it. This object can be achieved by studying some of the simpler structures in Nature which manifest recognizable evidences of vital activity.

There are in both the animal and vegetable kingdom structures which satisfy this requirement. They are present even in the human body, but as they are closely linked up with other structures their individuality cannot be differentiated from these with sufficient clearness.

In the lower animals and in plants there are structures consisting mainly of one type of cell, so that there is no difficulty in referring particular manifestations of vital activity to particular cells.

The more suitable of these activities for investigation purposes is that of movement, and in the lower animals and in plants there are structures composed of but one type of cell which exhibit movements, and these afford the opportunity of obtaining an insight into vital processes from which reliable deductions of great value can be drawn.

The principles on which the investigation is based.—Before setting out on such a quest it is necessary to have some fundamental principle or axiomatic truth by which accurate observations of facts can be interpreted. Biological science has advanced so far as to enable certain fundamental principles to be recognized. One of these is that a living cell is never at rest—the cell being engaged either in discharging energy in the shape of its functions or in renewing its energy.

So far as modifications of this activity can take place, they are dependent

upon the rate of renewal. The process on which this depends can be hastened or delayed. The time occupied by the discharge of energy, on the other hand, probably remains constant.

There are a great variety of agents which can hasten or delay the rate of the renewal of energy, and it is by the action of these agents that the various modifications of activity are brought about in all cellular structures. Taking, then, this view that the modifications of cell activity are due to variation in the rate of renewal of energy as the fundamental principle, and applying it to the interpretation of vital reactions, we obtain an insight into these reactions which affords an explanation of a great many manifestations which have hitherto been inexplicable.

Amongst elementary structures consisting of cells that exhibit activity, there are in plants certain structures which show movement. The parts concerned in producing the movement are found to consist of a congeries of similar cells. The study, then, of such movements enables the processes and structures to be referred to the properties of these cells. As they all possess the same properties, it is not difficult to recognize the part they play.

The functioning of plant cells.—For the sake of illustration, the hair on the leaf of the sundew plant (*Drosera rotundifolia*) may be taken. In this hair there is a series of cells concerned in the movements of the hair and another series concerned in secretion. In the following considerations of the movements of the hair the latter are not concerned, although the same principles are applicable to them.

The cells concerned in the movement are all identical and all possessed of the same faculties. If we start with the consideration of the functional activity of this structure when it is seemingly at rest, standing immobile, the question arises, What are the cells doing ? The cells which maintain the erect posture are living cells and therefore are never at rest. We can thus infer that the erect posture is maintained by the activity of the living cells, each of these cells being engaged at one time in discharging its energy, which takes the form of contraction, and at the other times being engaged in renewing its energy. We thus observe that these cells have within themselves the faculty of discharging their functions and of renewing their energy.

If an agent of an appropriate kind falls upon the leaf at the base of these hairs, such as a fly, then the hair bends down in a particular direction so that its apex is brought into contact with the fly.

In this act there can be observed the effects of a number of influences which have been commonly looked upon as functions of structures. There is shown, for instance, by this act evidences of *stimulation*, of *contraction*, of the *conduction of impulses* from the base to the apex. These functions are manifested by the cells on the side towards which the hair bent. The cells on the opposite side have acted in a manner which enabled this act to be performed and thus show the presence of *inhibition*.

It is to be noted, further, that all these functions were performed in an orderly manner. Certain cells were stimulated and contracted in a particular

sequence while other cells acted in an opposite manner. In this there is evidence of another function of the highest importance, namely, the function of *control*.

It is necessary to consider these different functions as exhibited by the hair of the sundew in some detail.

Stimulation.—The process of contraction started immediately after the fly alighted upon the leaf. Therefore the process of stimulation started in the cells which were touched. Seeing that the modification of the activity of the cell can only take place through the increase or decrease of the rate of recovery of energy, the effect of the fly alighting upon the leaf must therefore have hastened the recovery of energy of those cells which were brought in contact with the fly. As the evidences of stimulation extended beyond those immediately stimulated by the fly, the inference is drawn that with the discharge of energy of the cells at first stimulated an influence was exerted which hastens the rate of recovery of neighbouring cells. The inference can therefore be drawn that in addition to contraction the cells diffuse or discharge an influence which is capable of hastening the rate of recovery of neighbouring cells.

Contraction.—The fly having affected a series of cells which resulted in the bending of the hair, the inference can be drawn that a great many cells were stimulated to contract. As the hair was kept bent for a considerable time it is necessary to inquire how this was brought about. It has been assumed—and, as will be shown later, there are good grounds for the assumption—that the contracting function of a cell is but momentary, so that the maintenance of the bent position cannot be due to a long-continued contraction of the individual cell. The only other alternative is that the prolonged contraction is maintained by a continuous relay of cells passing into and out of activity.

Conduction.—As the evidence of contraction showed that the stimulation passed from the base to the apex of the hair, it is necessary to inquire how this came about. There being no other structure than these contractile cells it can only be through them that the influence reached from base to apex. In the description of stimulation it was reasoned that a cell received an influence which hastened the rate of recovery from neighbouring cells. The discharge of this influence as each cell contracts is quite enough to account for the contraction proceeding from base to apex, and there is no need to assume any other function or influence deserving the name of conductivity.

Inhibition.—It has also been assumed that in addition to agents which hasten the rate of recovery of the cell there are agents which delay the rate. There is evidence for this in the fact that while the hair bent, the cells on the other side must have opposed no resistance to the bending. Such lack of opposition indicates a diminished activity of these cells, and this can only come through an influence which is exercised by one cell upon another, by which the rate of the renewal of energy is delayed. So that in such an act as the bending of the hair we have evidences of influence which can at the same time hasten the rate of recovery of certain cells while the rate of recovery is delayed in other cells. These evidences of stimulation and inhibition occurring at the

same time are also shown when the stimulation exercised by the fly ceases, the hair then returning to the erect position. This can only have taken place by a cessation of activity or ' inhibition ' of the cells which had previously contracted, and an increased activity of the cells which had been previously inhibited. The only source for their ' inhibiting ' influence is the same as that from which arose the ' stimulation ' influence—the cells of this hair.

It may seem at first sight that a proposition which endows cells with a faculty to discharge at the same time functions or influences having opposite tendencies is illogical, but here we are not concerned so much with what seems illogical but with the recognition of facts, and on the interpretation of these facts in the light of the knowledge available. The undoubted possession by cells of these two functions and the results which they produce cannot be understood unless we recognize the presence of the influence of control.

Control.—Although the hair of the sundew is a lowly structure it is nevertheless very highly developed. In the course of this development it has attained that remarkable influence which pervades all normal vital activities, whereby such activities are regulated and designed to effect a definite purpose.

Again, it might be considered too great an assumption to endow cells with this particular quality, but as the study of vital activities proceeds reactions are observed to take place which can only be explained by the possession of such a quality. Here again it is necessary to bear in mind that all the evidences of a force in Nature are merely the effects which such force produces, and the presence of this influence of control is inferred from the purposive way in which vital activities are carried out. When later we come to speak of the loss of this influence of control additional evidence will be given for assuming its existence.

The cell impulse.—It has been very difficult clearly to recognize the nature of the force or influence which one cell exercises upon another. It has been described as the cell impulse, and in an attempt to comprehend more clearly what is meant by an impulse, the provisional conclusion come to is that it is nothing more nor less than those influences which result in stimulation and inhibition and control which have just been described. The supposition that there is a force which directly excites the cell to discharge its energy may not be necessary. In cells gathered together the discharge of energy is continuous, and it is conceivable that the discharge only takes place by some sort of chemical action when a sufficient store of material has been accumulated. At such a time the material may have become explosive. This, however, is still indeterminate at the present state of knowledge, but it is the view provisionally adopted here, on the principle that as Nature acts by the simplest means it is not necessary to explain phenomena by assuming the presence of other functions when they can be explained by functions already recognized. In the meantime the matter is left here, and an attempt will now be made to explain the nature of vital activities found in the study of the reactions of the human body.

CHAPTER XI

The Vital Activities in the Human Body

The contraction of muscle.
The structure and function of nerve cells.
The genetic system of the heart.
The functions of the sympathetic and vagus nerves.
The function of motor nerves.
The impulse, the nerve, and the reaction.
The influence of chemical agents.
Control.
Controlled and uncontrolled movements in the medusa.

In the previous chapter it was shown that the vital processes implied in the terms stimulation, inhibition, contraction, conduction, and control, could with certainty be referred to the functional activity of one type of cell. In more highly developed organs these processes became more specialized in some cells than in others, consequently the source of this process is not so easily traced. Nevertheless, all these processes seen in the more specialized structures are of the same nature and origin as those in the cells of the hair of the sundew.

The contraction of muscle.—In approaching the subject of the vital activities or reactions in the human body it will be best to begin with the most obvious, namely, that of contraction. Contractile organs are numerous, and productive of a great variety of phenomena both in healthy and diseased states. They are also extremely diversified in their constitution, and seemingly as diversified in the manner in which they perform their functions. This will be recognized when we find contractile organs present as varied as the voluntary muscular system, the capillaries, and the genetic system of the heart. Varied as are their constitution and function, an inquiry into the manner in which they perform their work shows that these activities are fundamentally the same. But before entering upon the description of cellular contraction, it is necessary to put out of mind the description of muscular contraction given in physiological textbooks, as practically all the observations on this subject have been made upon unnatural reactions in response to an unnatural stimulation. If there is one thing more conclusive than another which has come out of this inquiry, it is the error that has been fostered in imagining that the phenomena observed in clinical medicine could be explained by the results obtained by electrical stimulation.

Hitherto, in describing the muscle contraction, the muscle fibre has been adopted as the unit. A muscle fibre is described as usually being about one inch in length and $\frac{1}{500}$ of an inch in breadth. There is every reason for assuming that such a fibre is built up of a great many cells, although microscopic investigation may have failed clearly to differentiate them. The grounds for this view are partly on logical inference and partly on the observation of phenomena which can only be attributed to the independent activity of the individual cells.

In the condition called fibrillation of the muscle, fine movements are observed, sometimes only perceptible by a low-power microscope. These movements are manifestly not those of fibres one inch in length, but are due to movements in minute portions of the fibre which a reasonable inference can only attribute to the activity of individual muscle cells.

There are to be observed in clinical medicine conditions in which the muscle remains contracted for considerable periods of time, as in the prolonged contractions of voluntary muscle when engaged in prolonged efforts. Contractions of some duration are found also in involuntary muscle, as during the systole of the ventricle and the prolonged contraction of bowel and uterus. In addition to these normal manifestations there is the persistent rigidity of muscle found in the hardening of the abdominal muscles in visceral disease (the visceromotor reflex), in the contraction of muscles around an inflamed joint, and in the spasticity of various disturbances and injuries to the nervous system.

The manner in which these various forms of prolonged contraction are brought about can only be through the same form of activity which resulted in bending of the hair of the sundew and maintaining it in the bent position. As the contraction of each individual muscle cell lasts but a brief moment, as seen in fibrillation, the persistent contraction can only be due to relays of cells continually passing into and out of contraction, the immediate cause of this increased activity being the hastening of the rate of recovery after the discharge of energy.

Although the end result is the same in all forms of contraction, the origin of that influence or impulse which hastens the rate of recovery is different in different structures. While in the body there are structures which behave like a hair of the sundew where the influence or impulse originates in the cells that participate in the contraction, in other instances specialization of cell function has taken place, and this influence is exerted by cells specially evolved for the purpose, such as nerve cells.

The structure and function of nerve cells.—A characteristic of vital activities in the animal body is the modification of the structure of cells and the specialization of the functions. There are grounds for assuming a fundamental principle which can be expressed in the terms that all highly specialized functions are to be found in a rudimentary state in the more primitive cells.

As we have seen, the individual cell in the hair of the sundew possesses at least two distinct functions, the function of contraction and the function of discharging an impulse or influence which affects or modifies the activity of

neighbouring cells. Nerve cells have lost the function of contraction, while the other function, that of impulse formation and discharge, has become highly developed.

In the course of this development the cell has altered its character, so that different parts have become differentiated for specific purposes. There are fine threads or fibrils (dendrons) arranged in two groups, one at either end of a fibre (axon process) which connects them and the cell body (Fig. 10). The function of one set of fibrils is to receive the impulses from neighbouring cells. The effect of these impulses is the same as in the primitive cell of the sundew, to hasten or delay the period of recovery of the cell (or receptor fibril) which receives the impulse : this latter in its turn discharges energy in the shape of an impulse which travels by the fibre to the discharging fibrils. The function of these discharging fibrils is not only to act as a means of escape of the impulse, but these fibrils also give that peculiar character which enables the neurone to exercise an influence which either hastens or delays the rate of recovery of the cells with which it is in contact.

FIG. 10. Diagram of a neurone showing the four parts into which the primitive cell has become differentiated, each with its special function : 1, the receptor fibrils ; 2, the conducting fibrils ; 3, the discharging fibrils ; and 4, the cell body.

This description of the functions of the neurone or nerve cell is a reasonable inference arrived at from the consideration of the manner in which the primitive cell performs its functions. This view is supported by the consideration of the effects produced by the activity of nerve cells.

The view, then, is that the impulse or influence of the neurone or nerve cell is the same under all circumstances, its effect being either to hasten or delay the rate of recovery of the cells which are exposed to its influence. This is especially seen in the sympathetic and vagus nerves, but it will probably be found that this peculiarity of function is not limited to these nerves, but is common to every nerve, some having the one function and others having the other function.

The genetic system of the heart.—It is well known that the heart continues to beat when separated from its nervous connexions, and even when removed out of the body. This indicates that there are within the heart itself some structures which possess the power of stimulating the auricles and ventricles to contract in an orderly fashion. These structures are now recognized as the genetic system of the heart (see pp. 39-40).

The following is a brief description of the structure of this system so far as it has been revealed (see Fig. 2). In the auricle there is a collection of

cells of a type quite distinctive, having the characteristics neither of muscle nor nerve. A small mass of this tissue is found in the right auricle near the mouth of the superior vena cava—the sino-auricular node. No continuation of this structure has been recognized anatomically between it and the next portion of this system which is found in another mass of cells like those of the s.-a. node in the right auricle near the mouth of the coronary sinus called the auriculo-ventricular node. From this node there is continued a bundle which, passing into the ventricle, breaks up into innumerable fibres distributed to every part of the internal surface of the ventricles. The cells which constitute this bundle and its branches are of that irregularly rounded character which are recognized as Purkinje cells (see Fig. 16).

The investigation of this system has revealed that the initiation of the contraction of the normal heart arises from impulses generated by the cells of the genetic system in the auricle—chiefly those of the s.-a. node. During the contraction of the auricle the a.-v. node is stimulated to discharge impulses which result in the contraction of the ventricle.

The functions of the sympathetic and vagus nerves.—It is found that on warming the s.-a. node the rate of the heart increases, and on cooling it the rate decreases. The same thing happens when the sympathetic and vagus are stimulated experimentally, the former increasing the rate, the latter decreasing. It will thus be seen that the influence of temperature and of these nerves has the same result, both causing an increased activity or decreased activity of the cells of the s.-a. node. This can only be brought about by the modification of the rate at which recovery takes place in these cells after the discharge of energy. It can, therefore, be reasonably inferred that the impulses from these nerves are the same as the impulses discharged from the cells of such primitive structures as the hair of the sundew.

This way of looking at the matter has a very wide significance. It shows that living cells possess within themselves the faculty of discharging energy and of renewing it, and outside influences such as nerve influence act like heat and cold by modifying the rate of renewal.

It is also seen that the two opposed influences of increasing and decreasing the rate of recovery, which were possessed by each cell of the hair of the sundew, are exercised by two separate cells—those of the sympathetic and vagus.

The sympathetic and vagus nerves, which supply certain portions of the bowel, when stimulated cause a great increase in the strength of the contraction of the bowel, and because of this they are spoken of as being ' motor nerves ' to different portions of the bowel.

By the term ' motor nerves ' it is implied that these nerves convey the impulse which causes contraction, but from this way of looking at the matter, all that these nerves do is but to convey an influence which hastens the rate of recovery of the muscle cells. Like the heart the bowel can contract when these nerves are cut and their influence lost, *i.e.* the bowel possesses within itself those faculties which belong to all living cells of being able to discharge

and renew their energy, and there is no need to assume that these nerves have any other influence than to hasten the rate of renewal.

The function of motor nerves.—This view of the influence of nerve upon muscle is likely to be found applicable to the voluntary muscles. Here again the fact is recognized that when the terminal nerve to a voluntary muscle is cut the muscle cells exhibit the movements of fibrillation. This shows that the muscle cell possesses the power of discharging energy and restoring it. The only influence which is necessary to cause a contraction is to hasten the rate of recovery of a greatly increased number of cells, and to regulate the sequence of their activity.

The impulse, the nerve, and the reaction.—Looking at vital activities from the point of view given here, the conclusion can be drawn that the peculiarity of reaction, whether it is contraction, sensation, or secretion, is dependent, not on the character of the impulse nor on the nerves that convey the impulse, but on the function of the cells which ultimately receive the impulse, *i.e.* on the effector organ.

Nerves, therefore, exhibit no other property than that of exercising an influence upon the cells of effector organs, increasing, decreasing, or controlling their activity. Such terms, therefore, as afferent, efferent, sensory, motor, secretory, have no reference to the function of the nerve, but only to the structures over which it exercises an influence.

The same is true of the cell impulse. Beyond exercising the influences already described it has no other characteristic.

The influence of chemical agents.—There are a number of chemical agents which produce reactions similar to those of heat and cold, and to the action of the vagus and sympathetic nerves. These effects can be recognized in all sorts of structures. Certain chemical agents applied to the leaf of the sundew result in a contraction of the hair. Substances like adrenalin act in some respects like the sympathetic and heat by increasing the number of cells participating in the contraction. Other agents act by diminishing the activity of cells and preventing their action, such as atropine and curara. When all these effects are considered they are found simply to influence living cells by modifying the rate of recovery after the discharge of their function.

Control.—In pursuing the line of investigation suited to the discovery of the effects produced by natural forces, there are recognized certain reactions which can only be interpreted by assuming the presence of an influence which can be called ' control '. This was seen in the description of the purposive and effective manner in which the hair of the sundew bent in response to a stimulus. In this act of bending, while certain numbers of cells were stimulated to contract in a particular sequence, other cells diminished their activity in order to facilitate the bending of the hair. Such results can only be due to an influence which caused the different cells to act in these different ways. It was reasoned that the source of this influence could only be the cells themselves.

In the course of development some cells possess this quality in a higher

degree than other cells, and we find that these cells which possess it in a higher degree exercise their influence on other cells so that they control and regulate the activity of structures markedly different from themselves. This influence can be recognized in every normal structure which displays vital activity. So harmoniously do the different parts work together that its existence is scarcely recognized until the influence is lost. There are instances where the effect of control and the result of its loss can be observed in the human body. The relation of the voluntary muscle to its nerve is one of the most instructive. So long as a muscle is in connexion with a healthy nerve the activity of the muscle is restrained and regulated both when seemingly at rest and when engaged in active contraction. It not only causes the muscle to contract in a manner graded to suit the purpose of the moment, but it maintains an influence over the muscle when there is no call for its activity. In a quiescent state the influence which it exerts over the muscle causes relatively few of the cells to contract at any given moment, so that the muscle, though seemingly at rest, exhibits a certain degree of hardness or rigidity. This is due to a few muscle cells contracting in constant relays, the rate of recovery being delayed.

When the nerve to the muscle is cut a marked change takes place. In addition to its inability to respond to impulses from the brain, the muscle loses its firmness and hardness and becomes soft and flabby. The muscle cells are still supplied by nourishment, are still living, and therefore are continually in action, either discharging energy or restoring it. But they are no longer controlled so as to act together in a purposive manner. Each cell in the discharge of its energy not only contracts but stimulates the neighbouring cells to contract, and thus we find in the muscle a continuous movement which does not result in an organized or controlled contraction of the muscle on the whole but in unregulated contractions of individual cells (fibrillation).

In the description of the heart's movement it was shown that the cells of the sino-auricular node possessed the faculty of originating impulses which regulated the rate of the contraction of the auricles and ventricles. The whole genetic system represents the remains of the primitive cardiac tube by which the circulation is maintained in lowly developed animals. Before a definite tube is formed these cells possess in a high degree the faculty of contraction. When at an early stage some are removed from the body and placed in suitable nutrient fluid, they grow to form a thin sheet which exhibits a continuous movement. While in the body their growth is controlled, outside the body growth is uncontrolled, and their movements are not purposive.

This faculty of independent activity is retained by the cells of the genetic system, so that when the sino-auricular node is out of action, the auriculo-ventricular node can discharge impulses which produce ventricular contractions, and when this node is destroyed, the bundle can take up its function of causing the ventricles to contract.

The muscular system of the auricles and ventricles are outgrowths from the primitive cardiac tube and ultimately present anatomical characters

different from those of the cells of the genetic system. The muscle cells of the auricle and ventricle pass into fibrillation just like those of the voluntary muscle. It is presumed that this happens only when the influence of the genetic system is lost, but so far the intimate connexion between the genetic system and the muscle cells of the auricle and ventricle has not been made out.

When the heart contracts, the branches of the bundle which are visible in the exposed heart contract immediately prior to the contraction of the ventricle (Ishihara and Nomura). This is manifestly due to the passage of the impulse along the bundle, and shows that the impulse is not merely conducted along the bundle, but that it is by a process of stimulation and discharge of each cell that the impulse is passed on from cell to cell. This is the method of conduction described in the hair of the sundew.

When a portion of the bundle is cut away from its connexions and placed in suitable fluid, the same movements passing along the bundle can be seen to occur, showing a certain degree of co-ordinate activity due to its cells possessing this influence of control.

On the other hand, when portions of the cells of the ventricular muscle are taken out and placed in suitable fluid these cells exhibit movement of that disorderly kind characteristic of fibrillation, quite different from the co-ordinated movement of the bundle.

While these and other instances reveal the presence of this influence of control in individual cells and simple structures, this influence in complex organs becomes highly developed. It is on account of this influence that a number of structures different in function and in structure participate in that harmonious activity which results in the efficient functioning of a complex organ. While this can be illustrated in many ways, particularly through the influence of the nervous system, the matter is best illustrated by a simpler form of activity shown by the heart where both the significance of control and its loss can be appreciated.

The normal heart in the normal body exercises its function in supplying the organs of the body with sufficient blood for their several purposes. As these purposes are continually varying, requiring an ever-varying supply of blood, the heart accommodates itself to this varying activity in a remarkable manner. It is by the possession of this faculty that the heart's rate varies so greatly during the phases of bodily activity and rest, increasing or decreasing its output according to circumstances. This is brought about by the influence of the sympathetic and vagus nerves on the s.-a. node. These nerves are in contact with and are influenced by the afferent nerves of the organs whose activity is varying. So delicately balanced is the relation of these nerves that when one exerts its influence on the s.-a. node, the influence of the other is decreased. It is by their harmonious action that the heart is able to accommodate itself to the variations of bodily activity. In this complex way there is attained the same effect as was shown to occur in the movements of the hair of the sundew; at the time certain cells were stimulated the activity of others was decreased. Here is indicated the basis of ' reciprocal innervation '.

The delicate influence of the two nerves upon the s.-a. node is better realized when the influence of this node is lost. There are conditions in which this happens, and the contraction of the ventricles is maintained through the influence of the a.-v. node, as in the conditions called auricular fibrillation and flutter. When this happens the control exercised by these nerves is greatly altered or lost altogether, as shown by the response of the rate of the heart to effort. Sometimes there is little or no response, at other times the rate is greatly increased and bears no relation to the amount of effort. In auricular flutter the pulse rate may be found at 150 beats per minute, and this rate may be maintained when the body is at rest or when violent effort is made. In other instances, when at rest the rate may fall much below normal to between 40 and 50 beats per minute ; then on slight effort it may rise to 150 beats. Then, notwithstanding a great increase in effort the rate will not rise or vary in the slightest degree. This is seen in Figs. 7-9. Fig. 7 shows the rate of the pulse at 50 beats per minute when the patient was lying in bed. On getting out of bed the rate at once rose to 150 as in Fig. 9. Although the patient walked upstairs and down the rate never varied. On lying down after a few minutes the pulse gradually slowed until it fell to 50 beats per minute.

Not only does this show that the sympathetic and vagus nerves have a less perfect control over the a.-v. node than over the s.-a. node, but it will be seen in Fig. 8 that the size of the pulse beats, which indicates the quantity of blood thrown out with each contraction, varies with each contraction. Such variations as these never occur when the heart's contraction is under the control of the s.-a. node. It is therefore concluded that not only does the a.-v. node control the rate of the ventricle, but also controls the contraction of the ventricle. The control, however, lacks that fine adjustment shown by the s.-a. node.

In complete heart block the impulses for the contraction of the ventricle arise in the cells of the bundle. Here the functional control is completely lost so far as the regulation of the ventricular rate according to the requirements of the body is concerned, the ventricular rate being usually about thirty per minute while the body is at rest or undergoing violent exertion.

The importance of this way of looking at the matter lies in the fact that it is in a great measure on account of this loss of control that heart failure and death result. The very necessary knowledge of heart failure will never be understood until this aspect of the subject is appreciated.

This subject of loss of control is dealt with here in some detail, simply because it has been worked out more fully in the heart than in any other organ, but there is little doubt that the failure of other organs to function efficiently is brought about on exactly the same principles.

Controlled and uncontrolled movements in the medusa.—This principle of control, and the effects of loss of control, are well shown in the jelly-fish. The bell of the jelly-fish consists of several layers, of which there are two concerned in the movement, a network of nerve fibres overlaid by a layer of muscle cells. The nerve fibres are in connexion with the sensory bodies

situated at the margin of the bell. By means of these sensory bodies communicating, through the nerves, with the muscle layer, the movements of the jelly-fish are co-ordinated and regulated.

Mayer has shown that if a ring be cut out of a bell including the nervous and muscle layers, but freed from the sensory bodies at the margin, and placed in suitable fluid, the ring can be stimulated to contract, and a wave of contraction may be made to pass round and round the ring for weeks without ceasing. Such a movement effects no purpose, and it is not controlled or regulated in any way. The control of the sensory bodies over the movements of the bell is illustrated by the following experiment. If in place of separating the ring entirely, a narrow bridge is left communicating with the rest of the body and this ' circus movement ' started, a light touch upon one of the marginal sensory bodies immediately causes a cessation of the circus movement.

Romanes has shown that if these sensory bodies be removed the bell remains paralysed although responding to stimulation. Sometimes there suddenly starts an active shivering motion, which may last from a minute to half an hour. ' This motion ', Romanes says, ' is totally different from anything exhibited by the normal animal, and is evidently due to the various systems of muscle contracting without co-ordination.'

CHAPTER XII

The Vital Activities in the Human Body (*continued*)

Fluctuation.
Tone.
Centres.
The action of drugs and other chemical agents.
The manner of death.
The cell impulse and electricity.

Fluctuation.—Many years ago my attention was arrested by the manner in which the skin sensation varied or fluctuated in those patients whose skin showed hyperæsthetic areas. In the systematic investigation of cutaneous sensations at the St. Andrews Institute it was found that the description usually accepted was not in accordance with facts, and that the ideas of heat and cold spots, of the presence of touch corpuscles, and so forth, required to be reconsidered. Amongst the observations made was one which showed that heat and cold were felt from stimulation of limited areas of skin. Surrounding these limited areas the skin was insensitive to cold or heat, but after repeated testing it was found that the limited areas that were sensitive were most of them no longer sensitive, while the surrounding skin, which was previously insensitive, showed limited areas that had become sensitive.[1]

The inference drawn from this observation was that the cutaneous cells that were stimulated conveyed the impulses to the receptor fibrils of afferent nerves; that these cells were not all active at the same time, certain of them being active at one time and becoming inactive while other cells became active. To this alternating activity of cells we applied the term 'fluctuation', and we searched for evidences of this fluctuation in other tissues.[2]

The observation of the cutaneous capillary field afforded ample evidences that the capillaries behaved in exactly the same way. At one time a certain set of capillaries were in dilatation while others contracted; when the dilated ones passed into contraction the contracted ones dilated.

Recognizing, then, this aspect of vital activity, we applied it to the interpretation of phenomena in other organs, particularly in muscle, and we

[1] Waterston, D., 'The Sensory Activities of the Skin for Touch and Temperature', *Reports of the St. Andrews Institute for Clinical Research*, vol. ii. p. 123.

[2] Herring, P. T., 'The "Law of Fluctuation" or of Alternating Periods of Activity and Rest in Living Tissues', *Reports of the St. Andrews Institute for Clinical Research*, vol. ii. p. 133.

gradually came to recognize the explanation of muscle activity which has been given here.

When we reconsidered the whole question of cellular activity as illustrated by the description given of the movements of the hair of the sundew, we found, with the recognition of the manner in which the cell impulse acted, that this law of fluctuation was applicable to the normal activity of every structure, and that indeed so far as we can make out it is the only way by which the activity of organs can be regulated and graded to meet the varied requirements of the body.

It is not the purpose here to dwell at length upon the value of the recognition of the law of fluctuation in all cases, but the absence of its recognition has hitherto rendered impossible a clear understanding of how the cells of an organ played their part in the functioning of the organs, and in consequence it has been impossible to understand how their failure to function efficiently comes about.

It is not necessary to insist upon the great importance of understanding the nature of heart failure, yet this is a subject which has hitherto baffled all attempts at explanation. One phase of the subject was touched upon in dealing with the loss of 'control', another phase of the subject is seen in dealing with the law of fluctuation. The capacity of the ventricle to meet the varying needs of the body is due to its possession of this faculty. When the body is at rest, only a portion of the cells of the muscle are called upon to exercise their activity, and the cells which do so are continually varying. When effort is made, a greater number of cells participate in the contraction of the chamber. So long as there are a sufficient number of cells to produce an efficient contraction the circulation is well maintained, but when the number of cells is no longer sufficient for this purpose then the contraction becomes inefficient.

The agents of disease may cause a decrease in the functional activity of cells by their destruction or in other ways. The clinical evidence of this is to be found only by the signs of heart failure. This occurs only when the ventricles are compelled to contract with a force for which there are not enough cells to meet the demand. This forms the basis of that method of examination by testing the responses to effort which is of such great importance in the examination of the heart as of all other organs.

Another way in which this law of fluctuation is concerned in the response to effort is the state of the capillary field. When an organ is working quietly, a relatively small amount of blood is necessary. During this period only a portion of the capillaries are dilated. When the organ is called upon to perform more work, a greater number of these capillaries become dilated. The size of the capillary field is therefore of great importance for the work that the organ has to perform. In many diseased states, and particularly in what are called 'senile changes', the most conspicuous change is the diminution of the capillary field. When this happens to an organ like the heart, an efficient circulation may be maintained when the body is at rest and yet be

inefficient when effort is made, and in this way heart failure be brought about. It is necessary to grasp this method of activity, for hitherto the conception of the heart's contraction has been misunderstood because of the belief in the law of 'all or nothing'. This 'law' arose over fifty years ago at a time when the structures and processes concerned in the heart's contraction were very imperfectly understood, from the contemplation of the unnatural contraction produced by electrical stimulation. The belief of this law has prevented the understanding of many of the most common manifestations of the heart in health or in disease.

Tone.—What is meant by 'tone' has been difficult to understand. The origin of the idea of tone came about from the observation that voluntary muscles when resting showed a slight degree of contraction. It was also observed that when the nerve was cut this contraction disappeared, but what this meant escaped detection. With the recognition that cells are always active, either discharging their energy or renewing it, and that the rate of renewal may be so delayed that only a few cells may be discharging their functions at a given time, and with the recognition of the influence exerted by nerves upon the cells to which they are distributed, it can now be recognized that the condition to which the term 'tone' has been applied is the result of the contracting muscle cells. 'Tone' is therefore a form of muscular contraction and not in any sense a 'function' of muscle or any other cell.

Comment has been made on the relation of tone to the 'viscero-motor reflex', that is, the hardening of the muscles such as those of the abdomen in visceral disease, and the various other forms of muscle rigidity.

An identical condition can be produced in the heart. The relaxation of the ventricle during diastole is normally not complete. There are certain agents which render this relaxation less complete, so that during diastole the size of the chamber may be greatly diminished, so much so that little or no relaxation may take place, and the ventricular contraction may become ineffective because of this diminished relaxation during diastole. Other agents have the opposite effect and cause extreme dilatation of the ventricle with gradual loss of power of contraction.[1]

These varied results have been attributed to an increase or a decrease of tone, but they can be explained as due to agents which increase or decrease the number of cells in contraction during diastole, or, in other words, to agents which hasten or delay the rate of recovery of the muscle cells.

The significance of the recognition of tone as a reaction compels a reconsideration of this term, for it is in common use to describe phenomena which appear in structures that do not contract. The use of the expression increased or diminished 'tone' of a nerve or of this or of that 'centre', is indicative of the confusion of mind which is associated with the use of such terms.

Centres.—In the early stages of an investigation into a subject dealing with natural phenomena terms are employed for convenience at a time when

[1] Gaskell, W. H., Schafer's *Text-Book of Physiology*, vol. ii. p. 195.

the knowledge of their nature is very imperfect. Such terms, and the meaning attached to them, should only be looked upon as provisional and as representing the state of knowledge at the time that they were bestowed. With the advance of knowledge the terms and their meanings should be constantly revised. The failure to do this is bound, in course of time, to result in the employment of language which not only fails to describe the nature of the phenomena but may give a false impression of their nature.

This comment is illustrated by the use of terms in the various departments of medicine and has been sufficiently illustrated by the remarks upon the terms supposed to describe vital processes.

In all investigations phenomena are met with which at first are not susceptible of explanation. It may happen that the introduction of some fictitious or imaginary structure may give a plausible explanation, and in course of time such a term passes into common use and is accepted as indicating the existence of something very definite. Of many such terms ' centre ' is perhaps the most common in use. As employed to-day it merely affords a way out of a difficulty, and while helpful from this point of view it is absolutely deterrent to progress, inasmuch as it gives a false conception and leads to false descriptions of other phenomena.

There are a series of terms associated with ' centres ' which have really no existence. A ' pain centre ' is supposed to exist somewhere in the brain to which ' pain nerves ' convey what are known as ' pain ' or ' sensory ' impulses. Such terms convey the idea that there are impulses and nerves and a centre of this distinctive kind. This idea has led to the multiplication of a great many kinds of impulses, nerves, and centres, when, as a matter of fact, the impulses and the nerves which convey them have no such distinctive character, and there is in all probability no structure to which the term ' centre ' could be applied.

If we return to the consideration of the movements of the hair of the sundew, it was recognized that the influence of one cell could increase or decrease the activity of its neighbouring cells. These cells all discharge a particular kind of energy shown by their contraction. There are other cells in the hair which possess the function of secretion, and with the bending of the hair this secretion is greatly increased. It is reasonable to infer that the same form of influence which affected contractile cells also affected the secretory cells. But it would but confuse the matter to speak of this influence as a ' motor ' impulse in the one case and a ' secretory ' impulse in the other case.

There are many reactions in the human body which, though more complicated, are fundamentally the same as those occurring in the hair of the sundew. A calculus in the ureter can start up a series of reactions on exactly the same principle. Cells are stimulated into activity by the presence of the calculus, these discharge impulses which affect neighbouring cells, and each cell produces a reaction characteristic of the functions of the cell. In this way we get a series of manifestations of reactions such as pain, nausea, vomiting, sweating, and so forth. As pain is the most clamant symptom in such

reactions, it has given rise to the common belief that this is an evidence that the ureter must possess ' pain ' nerves and give rise to ' pain ' impulses. This is not a correct way of looking at the matter, for one would be as justified in stating that the ureter possessed ' nausea ' nerves, ' vomiting ' nerves, ' sweating ' nerves, and gave rise to ' nausea ' impulses, ' vomiting ' impulses, and ' sweating ' impulses. As there are a great many more reactions than these, one would be forced to endow the ureter with a great many other kinds of nerves and impulses.

Another line of argument brings us a little nearer perhaps to the understanding of what this structure is which is camouflaged by the term ' centre '. The fundamental proposition with which this investigation started was that living cells were never at rest, being engaged either in discharging their function or in renewing their energy. It follows from this that the function of no individual cell is devoted to a distinctive sensation such as that of pain, and there can be no nerve or centre specialized for the production of pain. If there were specialized cells or centres of this sort, pain would either be a constant phenomenon or, in other instances, its cells would not discharge their function in the course of a life-time. The question arises, how can a sensation like pain be produced ? If we observe what happens in any complex reaction such as vomiting, we find muscles employed, but these muscles are not specialized for the purpose of vomiting. They are never at rest, but it is only when cell activity results in a particular combination of muscle contraction that vomiting is produced. At other times they are engaged in such acts as respiration or swallowing.

The eye perceives a great many shades of colour and the ear many shades of sound, but no one would suggest a ' centre ' for each of those shades of colour or sound. It is manifest that the recognition of these shades is the result of a combination of cell activity on the same principle as that which resulted in vomiting.

Such sensations as pain, therefore, are not the result of specialized impulses or nerves or centres, but they arise by a particular combination of cells which are normally engaged in other functions.

In the description of the structure of a cell it was shown that particular parts had become specialized for the performance of particular functions; that for the reception of the incoming impulse and the discharge of its own impulse the receptor and discharging fibrils of the neurone were specialized. The character of the impulse, whether it increased or decreased the rate of recovery in the neighbouring cell, depended upon the discharging fibrils. As both kinds of fibrils are influenced in the usual manner of cells by having the rate of their recovery hastened or delayed, it is by the participation of these parts of the cell and not by the cell body that the various complicated reactions which have been attributed to a ' centre ' are brought about.

It is not possible at present to explain further what is meant by a centre, for this is one of those problems which requires for solution a far more intimate knowledge of vital processes than is at present available, but there is no reason

for assuming anything mysterious about the matter, for it is almost certain that the result is brought about by a combination of those elementary forms of activity which have been described.

The action of drugs and other chemical agents.—(Drugs, poisons, toxins of disease.) If there is any department of medicine whose progress is hampered more than another by the lack of a proper conception of vital activity, that department is therapeutics. The taunt that has been so long levelled against the doctor is as true to-day as when it was first uttered, that he gives drugs whose actions he does not understand for conditions of which he is ignorant. There is every prospect of the state implied in this taunt continuing so long as the attitude towards medicine which is dominant to-day persists. The views expressed here do help materially to provide an understanding of the manner in which drugs and other agents act. Darwin, in his observation on insectivorous plants, showed that the hair of the drosera bent down and discharged its secretion in response to certain chemical agents placed on the leaf. An extremely minute quantity of a nitrogenous solution would cause this reaction while it was negative to solutions of gum arabic, olive oil, etc. These agents in producing this reaction acted in the same way as the stimulus which resulted from a fly alighting on a leaf, *i.e.* by shortening the period of recovery in some cells and delaying the period of recovery in other cells.

There are reasons for suggesting that it is in one of these two ways that all other agents act, whether drugs or the toxins of disease. The numerous and varied manifestations which are the result of such agents are merely dependent upon the functions of the structures affected and whether their activity is increased or decreased.

The superficial appearance of a reaction gives no indication of how the disturbance started. Atropine in sufficient quantities given to an individual increases the heart-rate, and at one time it was looked upon as a cardiac stimulant. Digitalis, on the other hand, diminishes the heart-rate, and it was therefore looked upon as inhibiting the heart. When these antagonistic reactions are analysed it is found that atropine is not a stimulant, as it acts by delaying the recovery of the vagus; while digitalis is not an inhibitory agent, as it acts by hastening the recovery of the vagus.

These drugs do not act on the same structures. A neurone of the vagus possesses its receptor fibrils and its discharging fibrils. In the discharging fibrils lies the structure which gives rise to that peculiar effect of the impulse whereby it causes a delay in the recovery of the cells on which it acts. Atropine acts upon these fibrils so that they gradually cease to respond to a stimulus on account of the increasing delay in their recovery.

The receptor fibrils, on the other hand, can have their recovery hastened and delayed in the same way, and the action of digitalis is to hasten the recovery of the receptor fibrils so that the vagus becomes much more receptive to impulses from without.

There are conditions in which reactions of a very striking character occur

in response to agents of this class. One of these reactions is called anaphylaxis. This is a condition which nobody understands, and the name is given because it is supposed to describe some form of activity, but it is not known how to set about finding its nature. There are other similar, if not identical, forms of activity. One of these is seen in the response of the heart to digitalis. For over 100 years it has been recognized that digitalis has little or no effect upon a great many hearts, while there are some hearts upon which the effect is very great. An individual may at one time show no reaction of the heart to medicinal doses of digitalis. At another time the same doses of the drug may cause a rapid decrease of the rate from 120 or 130 beats per minute to 50 or 40 beats per minute. Occasionally when the heart is thus reduced in rate, and still under the influence of digitalis, the rate may suddenly increase to 70 or 80 beats per minute, and increased doses of digitalis then have no further influence in slowing the heart. Such a reaction as this, which I have observed on many occasions, is quite beyond the possibility of explanation by means of the conceptions of to-day. But according to the view set forth here the matter can be explained, if not completely, at all events sufficiently to give a remarkable insight into vital processes.

It has already been pointed out that so long as a voluntary muscle is under control of its nerves, the muscle activity is not influenced from any other source than through the nerve. When this influence is lost the muscle is influenced in other ways and passes into fibrillation. It was also shown that the sino-auricular node (or perhaps more correctly the genetic system in the right auricle) controls the movements of the heart, and when its influence is lost the a.-v. node then regulates the movements of the ventricles. When this happens, that fine control whereby the movements of the heart respond to the requirements of the body is lost, and the a.-v. node becomes extremely susceptible to influences which either increase or decrease its activity. When its activity is increased, as shown by a greatly increased rate of the ventricle, digitalis has the effect of rapidly reducing this rate in the manner just described. If, as not infrequently happens, the s.-a. node is suddenly restored to activity, it discharges impulses at a rate of between 70 and 80 per minute, to which the ventricle at once responds. An increase of digitalis when this happens may have no effect whatever upon the s.-a. node. The seemingly mysterious response of the heart to digitalis is thus seen to be dependent upon the nature of the disturbance which deranged the heart's action.

This illustration of the action of digitalis is merely given to indicate that a new field of observation is opened up.

Chemical agents, including the toxins of disease, while disturbing the functions of cells in the manner described, also produce effects which lead to an alteration in structure, but this is outside the present purview. It may be said, however, that this way of looking at the matter also throws light upon these structural changes, a field which stands much in need of investigation, and we have gone so far to see that the line of investigation pursued here

will be equally fruitful in dealing with the origin and nature of the structural changes and the effects they produce.

The manner of death.—It is a curious matter that notwithstanding that the main efforts of the medical profession are devoted to the prevention of death, yet nowhere is there to be found any description of the manner in which death is brought about except in terms so vague as to throw little light upon the subject. Death takes place when the ventricles cease to throw out blood. While they can perform their function the tissues of the body are supplied with nourishment and they live, but when deprived of blood they speedily cease to function. Whatever may have led up to the heart's failure to function, this is the ultimate factor.

This failure to function may be brought about in different ways, but in the end the failure is due to a delay in the rate of recovery of certain cells.

The best illustration is to be found in the death that supervenes on the cessation of respiration. The cessation of respiration is itself brought about by the failure of the same process.

On one occasion, while I counted the pulse of a patient under chloroform, the breathing stopped on account of the falling back of the tongue, and the pulse rate gradually fell from 60 beats to 20 beats per minute. On breathing being started the pulse at once rose to over 60 beats per minute. This slowing of the heart-rate was entirely due to a delay in the rate of recovery of the cells of the s.-a. node. This was brought about by the action of the impure blood. It is not necessary here to discuss whether it was due to the absence of oxygen or the presence of some chemical agent.

In experiment, the death which follows the cessation of respiration is brought about by the same process. In the earlier stages of asphyxia the rhythm of the ventricles may become irregular, due to the dropping out of ventricular systoles or heart-block on account of a delay in the recovery of the cells of the a.-v. node. When the ventricles ultimately cease to beat, death follows. Not infrequently for some time after the output of blood from the heart has ceased, portions of the right auricle continue to beat, hence the auricle is said to be the *ultima moriens*. This is due to the fact that certain cells of the genetic system are still active, but those that convey the impulses from them to the ventricle are no longer susceptible to stimulation on account of the delay in their recovery.

On these lines other forms of death can be accounted for.

The cell impulse and electricity.—From the study of the effects produced by the agent or force called the cell impulse, all the varied forms of vital activity or reactions can be attributed to the various combinations of the influence exerted by this agent. These influences are themselves few in number, producing an increase or a decrease in the rate of recovery of the cell after discharging its energy and the influence of control.

Another feature of this impulse is that its effects do not extend beyond the cell with which it is in immediate contact. This is a point which has not been grasped, for attempts have been made to compare the conduction of impulses

in structures widely different in constitution, such as the conduction in nerves with the conduction in the auriculo-ventricular bundle. In the one case the passage of the impulse was measured through one or at the most through two cells, in the other case the impulse passed from one cell to another through an incalculable number, so that there can be no grounds for comparison of the conductivity of such structures.

The cell impulse is thus seen to be a force peculiar and distinct from any other known force. It is generated by cells, and it only acts upon cells in the manner just described. The other force, electricity, which has been used as a substitute, has totally different qualities. It is conducted by any structure that is mechanically suited to the purpose. It acts upon cells in but one way, by stimulating them to activity, and there is a total absence of that influence of control which is so essential to the functional efficiency of organs. That information of value can be derived from electrical stimulation no one can deny, but there is little doubt that the failure to appreciate the difference between this unnatural stimulation and the natural has not only obscured the nature of vital activities, but has led to their misinterpretation.

CHAPTER XIII

The Classification of Symptoms

The object of a classification.
The basis of a classification.
The principles of classification of the symptoms of disease.
Classification and investigation.
The application of classification to investigation.

The object of a classification.—The multiplicity of symptoms has already been a subject of comment. As clinical medicine continues to split into branches, due in a measure to the recognition of new symptoms and new methods for their detection, the mass of symptoms is continually growing. If to this mass there is added the reactions — which are in nature identical with symptoms—obtained in other branches of medicine, as bacteriology, pharmacology, and physiology, there results a great mass of phenomena which is so chaotic that it is not possible to appreciate fully their nature and significance or their relation the one to the other.

The accumulation of a mass of confused details is characteristic of the early stages of all investigation of natural phenomena. A time comes when it is realized that the mere addition to this mass does not help progress, but hinders it. If progress is to be made, some concept must be introduced which will render the phenomena easier of comprehension, and which will act as a guide to further research.

The basis of a classification.– To obtain such a concept has been one of the objects of our enterprise at St. Andrews. We adopted the proposition that while the phenomena in nature may be infinite in number, the laws governing their production are few. As the symptoms of disease and the various reactions obtained by experiment are phenomena in nature, it was assumed that the laws governing them would be few in number. In the search for these laws it was surmised that some intimate connexion might be found between the phenomena which would enable them to be classed in groups according to their affinity in nature.

In this search the following train of reasoning was taken as a guide. In the inquiry just described it was shown that in the production of symptoms and reactions there were present two factors, namely, living structures and vital processes. It was assumed that the laws might be discovered by a more intimate knowledge of these two factors.

It was found that the structures concerned in the functioning of organs were fairly well known, but there was a very considerable number of them. Moreover, in looking at the part they played in the production of any reaction, it was found that, however simple the reaction might be, more structures than one took part in its production. The possible combination of structures acting together was so great that it was evident that no feasible classification could be based upon the structures engaged in the production of symptoms.

The other factor, vital processes, offered a different prospect. In the description of the results of our investigations into the vital activities of cells, it was shown that they were dependent upon the accumulation and discharge of energy. It was also shown that the manifold variations were dependent upon the rate of renewal of energy after its discharge. The rate of renewal varies through influences which increase or decrease it, and which regulate the activity of cells collected together to form an organ.

As it is only in these three ways that vital processes act, all symptoms and reactions can be grouped according to which of these three ways the disturbance arose which produced them, and thus we are able to classify symptoms in the manner given in the following table. In this table the symptoms which are due to structural changes are not included, and the terms ' impulse ' and ' conduction of impulses ' are employed in the sense described in the preceding chapters.

The principles of classification of the symptoms of disease.—The following are the propositions on which a scientific classification of symptoms is based :

All forms of vital activity are due to the functioning of living cells.

Living cells are never at rest, but either discharging energy or renewing it.

Modifications of cell activity can only take place by hastening or delaying the renewal of energy.

When cells are grouped to form an organ their activity is regulated and controlled.

Cells, in discharging energy, exhibit their peculiar function (as contraction, secretion, sensation), and at the same time discharge an impulse.

The impulse exerts an influence on neighbouring cells which acts by increasing or delaying the period of renewal of energy.

Certain cells exercise a controlling influence over other cells. The functional activity of organs is regulated and controlled by structures other than those which are engaged in discharging the function of the effector organ. These structures with the effector organ constitute the elements of the reflex arc.

Symptoms are due to a disturbance of some element of the reflex arc. Such disturbances result in :

1st. An increase in the number of impulses.
2nd. A decrease in the number of impulses.
3rd. An interference with the generation or conduction of impulses.

These results give rise to three different kinds of symptoms or reactions.

1. An increased activity of an organ.
2. A decreased activity of an organ.
3. A cessation of activity, or an ill-regulated, or unregulated, or purposeless, or disorderly activity of the organ.

The application of classification to investigation.—Looking at symptoms as presented in the foregoing classification, a principle emerges which guides investigation, namely, the principle of the reflex arc.

The application of the principle of the reflex arc in investigation has for its object:

1. The detection of the part or element of the reflex arc whose disturbance causes the symptom.
2. The detection of the nature of the disturbance—that is, whether due to increased activity, or decreased, or interference with control.

This leads to the recognition of the seat of disturbance and its nature.

The next step is the consideration of the agent which produces the disturbance, which is not considered at present.

This classification may seem at first to be of little practical value, and some difficulty may be experienced in seeing how it can supply the requirements of a classification which would at the same time group symptoms and reactions according to their affinity in nature and act as a guide for further research. When it is reflected that there is no clear conception of the manner in which agents of disease or drugs act in producing their effects, such actions being more or less a mystery and such descriptions of their actions being mere conjecture, the recognition that they act in the simple manner indicated here enables them to be grouped in the first instance according to whether the rate of recovery is increased or decreased or the influence of control interfered with.

As an illustration, the symptoms or reactions which indicate the loss of control as shown by disorderly activity may be taken. When the nerve to a muscle ceases to act upon the muscle, the muscle cells exhibit that form of ineffective activity called fibrillation. The recognition of this symptom, and its disorderly character, lead to the conclusion that the source of the disturbance is not to be found in the muscle but in the structures which regulate and control its activity. In affections of the heart the auricle is frequently found to be in fibrillation. According to the principles laid down here it would be futile to search for the cause of that disorderly activity in the muscle of the organ; search, to be successful, must be made into the structures which control the muscle, or which conduct the influence of control from its source. This we now know is to be found in the sino-auricular node and portions of the genetic system within the auricle.

It is recognized that the s.-a. node controls other structures than the auricle, chief of which is the a.-v. node and through it the ventricles. When the effects which result from the uncontrolled a.-v. node are inquired into, they are found

to be of a very important kind. It is as a result of uncontrolled activity that the ventricle acts in a way which it does under no other circumstances, often resulting in the inefficient performance of its functions, leading to a particular form of heart failure and to death.

In looking at the matter from this point of view it is found that the uncontrolled auricle is a matter of little importance compared to the importance of the uncontrolled a.-v. node.

The study of the a.-v. node under these circumstances has opened up a new field of inquiry and has afforded an insight into vital activities which has not been obtained by the study of any other structure. To a great extent the conceptions of vital activity, given in the preceding chapter, are drawn from the study of the a.-v. node when disturbed in the three different ways alluded to.

There are a great many other instances of disorderly activity which have hitherto baffled all attempts at explanation. The most striking of those are seen in the nervous system, as in delirium, insanity, epilepsy, migraine, and the different forms of paralysis. In paralysis very little consideration has been given hitherto to the behaviour of the structures which have been deprived of the controlling influence. The line of inquiry pursued here will certainly throw a good deal of light upon paralysed structures. This will be realized when it is borne in mind that these structures are composed of living cells supplied continually with nourishment and showing the characters of living cells, never being at rest but continually engaged in discharging energy or in renewing it.

Further illustrations of the value of this classification will be given, but particular attention is drawn to the classification of irregular action of the heart on page 107.

CHAPTER XIV

The Rôle of the Capillaries in the Production of Symptoms

The capillary as an organ.
The regulation of capillary activity.
Nerves and the capillaries.
Capillaries controlled.
Capillaries uncontrolled and failure of the circulation.
The capacity of the capillary reservoir.
The cause of the failure of the circulation due to the capillaries.
The obliteration of capillaries.
Effects produced by obliteration of the capillaries.
The cause of capillary obliteration.
The obliteration of the capillaries in the eye.

The capillary as an organ.—In the past, authors were wont to adopt for a title for their books Diseases of the Heart and Aorta, under the impression that the heart and aorta were the most important parts of the circulatory system. On the other hand, I expect that such a book as this should be described as Diseases of the Capillaries of the Heart and Blood-Vessels, for the heart and the blood-vessels are dependent for their integrity upon their capillaries, and their defects lead to grave results.

The capillaries must be considered as organs, and subject like all organs to influences which regulate and control their activity. We have seen that they obey the law of fluctuation and so vary their activity to meet the requirements of the body. Like all other organs they are controlled and subject to conditions which increase or decrease their activity, and they are liable to have their activity suspended, and of all organs of the body they are the most susceptible to the agents of disease. In this we see that they must have a system which regulates and controls them, but at present it is not clear what this is. In other respects they behave like organs.

The regulation of capillary activity.—In the discussion of the parts concerned in the conduction of impulses conduction was shown to take place by neurones and from cell body to cell body. The latter occurs in plants, in the lower animals such as the sponges, and in the auriculo-ventricular bundle. It is probable that the same form of conduction occurs in capillaries, and that their activity is regulated in the same way. This view finds support in the fact that capillaries are, of all organized structures, the most primitive and most easily developed, so that we find them growing in wounds, as in granulations, before it is possible

for nerves to accompany them. So far as we can see, the capillaries in the granulations have the power of dilating and contracting.

Krogh [1] has shown that capillaries are not tubes whose function is merely to act as channels for the blood flow. In groups of capillaries supplied by the same arteriole, the capillaries do not all act in the same way at the same time, for some dilate while others contract. Obviously then, the signs of activity cannot be due to arterial pressure. In one experiment, Krogh demonstrated that the whole length of the capillary from an arteriole to a venule is contractile, and when contracted it cannot be forced open, though it can open in response to a suitable stimulation.

He shows further that there is a direct influence exerted by the cells of an organ upon its capillaries. During muscular action large numbers of capillaries become visible and dilated. Shortly after the muscle returns to rest most of the capillaries disappear, only a few of them remaining open.

The source of stimulation to which the capillaries respond seems to be the impulses discharged by the contracting muscle. This is also the source of the impulses which are conveyed into the central nervous system by afferent nerves (p. 93).

Nerves and the capillaries.—It is not necessary to dwell at length upon the increased and decreased activity of capillaries. The phenomena of dilatation which occur in flushing indicate that nerves act upon the capillaries in the same way that the sympathetic and vagus act upon the heart, by moderating the regulating system, whatever that may be.

Seeing that the suggestion is made that the cells of which the capillaries are built up possess this regulating influence, it might seem an unnecessary refinement to conclude that the nerves do not directly control the capillaries, but the idea I have in mind is that the capillaries possess within themselves this power which, though susceptible to nerve influence, can act independently of it, so that there may be dilatation of the capillaries without nerve stimulation.

The reason that I make this suggestion is that it helps us to understand some very important phenomena which have so far escaped notice.

Capillaries controlled.—In watching people who are dangerously ill, gradually sink, and die, remarkable differences are found in the behaviour of the capillary system. Many people, as the end approaches, suffer from heart failure of that kind which occurs in auricular fibrillation, and show evidences of stasis in the peripheral vessels, particularly in the venules, by cyanosis of the lips, of the cheeks, of the ears, and other parts. This implies that the blood has passed through the capillaries and lost its oxygen, but the force of the heart-beat is so nearly spent that the blood tends to accumulate in the veins. At the post-mortem examination of such patients the veins are found engorged with blood.

Capillaries uncontrolled and failure of the circulation.—If, on the other hand, we watch the oncoming of death in patients dying with malignant infections, such as puerperal fever, within a day or two of the infection,

[1] Krogh, A., *Anatomy and Physiology of Capillaries*, 1923.

remarkable disturbances are to be witnessed in the peripheral circulation. The radial pulse becomes small and thready—the eyes are sunken, and the face is drawn and pallid. When death occurs there is no evidence of stasis of blood anywhere. At a post-mortem examination the veins and the heart are empty. The question arises, ' Where is the blood ? '

The capacity of the capillary reservoir.—Dale and Kellaway[1] in their experiments upon histamine shock have shown that in this condition there is great dilatation of the capillaries, particularly of those of the limbs, and the blood has all accumulated there.

From his experiments Krogh has reckoned that the capillaries, when fully dilated, are capable of containing all the blood of the body many times over.

The cause of the failure of the circulation due to the capillaries.—The explanation which I suggest is that certain toxins have a peculiar affinity for certain structures of the body. It is by symptoms produced in these structures that we are able to differentiate the different fevers, such as measles, scarlet fever, typhoid fever, pneumonia, etc. In like manner the toxins of the malignant infections have a peculiar affinity for the structures that regulate the functions of the capillaries, and this regulating function being abolished, the capillaries dilate and accumulate within them an undue proportion of the blood.

This view brings this type of ill-health into relation with all others, its gravity consisting in the fact that the toxin acts on the structures necessary for the maintenance of life, while the toxins of non-malignant fevers act upon structures whose integrity is not necessary for the maintenance of life.

The obliteration of capillaries.—The functional activity of the cell is dependent upon a supply of suitable nourishment. It is a matter worthy of consideration to find what happens to a cell when its blood supply is defective or ceases. No doubt the functional capacity is impaired in the first instance, and when the supply ceases certain changes are bound to take place. The living cell is never at rest, and the chemical processes within the cell are bound to be modified; and although it is not possible to describe these modifications, we can detect the results. They are twofold. The cell alters in structure to constitute fibrous tissue in which lime salts may be deposited, or it may result in the disappearance of the cell, leaving nothing behind. This latter result is seen in the skin.

If we watch the changes that take place with advancing years we will discover many features due to the impairment of the blood supply to the different organs and tissues, and it is possible to recognize that the cause of this diminished blood supply is the disappearance of the capillaries. We have but to look at the hands of some elderly people to find the skin thin like tissue-paper. When a cut is made through the skin, in place of that free oozing of blood which is so characteristic of the bleeding of a wound in

[1] Dale, H., and Kellaway, C. H., ' Anaphylaxis and Anaphylatoxin ', *Philosophical Transactions of the Royal Society*, B, vol. 211, 1923.

the young, there is little or no oozing from the capillaries, but a small jet from an artery, or a slight trickle from a vein. The vast capillary field has become greatly diminished, and what takes place in the skin occurs in every organ of the body, and if search be made, plenty of evidences of the same kind as that found in the skin can be obtained.

While I am not qualified to speak of the microscopic appearances of the tissues in which the capillaries have disappeared, there is abundant clinical evidence of the effects produced by the obliteration of capillaries.

Effects produced by obliteration of the capillaries.—There are some tissues more liable than others to the degenerative changes that follow the obliteration of capillaries, although there is no organ or tissue in the body which is exempt. The earliest changes appear in those structures with the poorest blood supply, such as the valves of the heart, probably the elastic coat of arteries, and at the margins of the cornea, where they appear as the arcus senilis.

If one listens carefully to the sounds of the heart in people over fifty years of age, systolic murmurs will frequently be heard either at the apex or base of the heart. At the post-mortem examinations the edges of the mitral and aortic valves will be found thickened and slightly shrunken. Such changes in themselves scarcely embarrass the heart and are of little or no significance so far as the health of the individual is concerned. But sometimes these changes in the case of the aortic valves extend into the aorta and cause a sclerotic thickening. If this sclerotic thickening should extend to and embrace the mouths of the coronary arteries these become rigid and narrowed. In the normal action of the heart there is required a very great increase of blood to the heart muscle when bodily effort is made. The coronary artery is normally very distensile to meet this increased supply of blood. When, therefore, the orifice of the coronary artery becomes rigid and narrow, the heart muscle does not get enough blood when effort is made, and shows signs of its embarrassment by the production of pain (see p. 327 and Plate I.).

Similar sclerotic changes can occur in other portions of the heart, and a part which is not infrequently affected is that immediately under the aortic valves. The sclerotic changes which occur here may involve the auriculo-ventricular node and bundle, and heart-block results (see Observation, p. 259). I suggest that similar changes may take place in the neighbourhood of the sino-auricular node, and impair, or destroy, its function, with the result that the auricle passes into fibrillation, as it is in elderly people that the condition is most frequent (see Observations 64 and 65).

The capillary obliteration may affect the walls of arteries, and lead to such degenerative changes that the arterial lumen may be blocked. When this affects the coronary artery the blood supply of considerable areas of the ventricle may be stopped and the muscle be changed into a mass of fibrous tissue, seriously impairing the functional efficiency of the heart. This inefficiency is shown when a call is made upon the heart to supply more blood

during effort, and the exhaustion of the muscle is indicated by the production of pain.

As no organ of the body is immune from the effects of this capillary obliteration, its evidence is to be found in the impaired function of every organ. These evidences are extremely instructive as revealing the part played in the economy of the different organs and structures.

Kidney function is impaired, and such impairment is often accompanied by the appearance of albumin in the urine. Pancreatic function is impaired and shown by glycosuria. The muscles of the body are impaired, shown by weakness and pains in the legs after walking, in the arm after writing, and in the back after sitting erect for a long time. Many parts of the brain are affected—one part in one individual and another part in another, so that we get a great variety of evidences of the impairment of control functions in the elderly, and so on with regard to every organ in the body.

The cause of capillary obliteration.—In my young days some philosophic physicians endeavoured to establish a doctrine that the latest developed parts were more liable to be affected by disease. This was not carried far enough to be of much value in unravelling the mysteries of disease. I have been trying a modification of this doctrine to see how it would work. This modification is that the organized structure most readily developed is the one most liable to destruction. Of all organized structures of the body the capillary is the one most rapidly developed, as shown in the granulations of a wound. It is the most easily destroyed, for if a healing wound be slightly touched with sulphate of copper the only structures destroyed are the capillaries.

There seems to be a great susceptibility for certain cells to be changed into fibrous tissue. The cells of the liver become fibrous in response to a number of agents such as the salts of manganese, the silicates, mercury, and even to tar. Lorrain Smith tells me that biliary cirrhosis may readily occur in a rabbit after rubbing tar behind the ear. I mention this merely as a suggestion that many diseased conditions may possibly arise from the obliteration of capillaries.

This obliteration has other far-reaching effects; the tissues that are poorly supplied with blood are more liable to microbic infection. In an amputation of a limb where there is a long flap poorly supplied with blood, should suppuration take place, it may be found that, while the sutures of the short flap are soundly healed, the sutures of the long flap are converted into ulcers. It is on the valves that have been damaged and become cicatrized that the microbes of malignant endocarditis are apt to settle, and possibly it is for the reason that they are so inaccessible to the blood stream that the valves are the foci of infection.

In certain tissues which have been inflamed, after the inflammation subsides, the cells of the tissue are found to be replaced by connective tissue fibres. Such scar tissue is very sparsely supplied with capillaries, and the question arises, Are these connective tissue fibres the result of the obliteration of the capillaries? We have seen reasons for suggesting that the capillaries

are the most easily destroyed of all organized structures. The agents which produced the inflammation may have so injuriously affected the capillaries that they die.

The obliteration of the capillaries in the eye.—In applying the principles of research here enumerated, we seek for an explanation of the symptoms produced in one structure by studying other structures which are more readily explored. The morbid processes which occur in other organs occur in the eye, and as this organ affords the opportunity for the observer to witness the progress of disease processes, we find the examination of the eye of the greatest service.

Maitland Ramsay has favoured me with the following note on the obliteration of capillaries and the effect they produce in certain regions of the eye:

"The optic nerve is a good illustration of the truth of the statement that the functional activity of an organ is determined by its supply of capillaries. Those fibres of the nerve which go to the macula are distinct from the others, and are called the papillo-macular bundle. They form a cylindrical group in the axis of the nerve until it approaches the eyeball. They then change both their shape and position and reach the periphery of the nerve, to be distributed in the temporal aspect of the optic disc as a wedge-shaped bundle, the apex of which is directed towards the central blood-vessels. These papillo-macular fibres are very abundantly supplied with blood on account of the extremely fine capillary mesh-work which surrounds them; consequently, they are the most liable to nutritional derangement when a toxic agent affects the optic nerve. In tobacco amblyopia, for example, the ophthalmoscope usually reveals pallor of the temporal portion of the optic disc, *i.e.* of the area of the disc corresponding to the distribution of the papillo-macular bundle. This ophthalmoscopic appearance is sometimes described as partial atrophy of the optic disc, but that is inaccurate, because in the majority of cases the normal appearance of the disc is restored when the patient recovers. The pallor is therefore much more likely to be due to anæmia of the papillo-macular bundle from interference with its capillary blood supply. On the other hand, when the capillaries of the papillo-macular bundle are destroyed, as takes place in certain forms of retro-bulbar neuritis, the pallor of the temporal aspect of the optic disc remains permanent, and the atrophy extends to other parts of the nerve.

"The retinal blood-vessels are not distributed to the macula itself. The layer of cones forming the macula derives its nourishment from the vessels of the choroid, that is, from the chorio capillaris, which in this region forms a particularly fine mesh-work. Of the whole retina, therefore, the macula is the part most abundantly supplied with capillaries. It is also the most vulnerable portion of the retina, and any interference with its blood supply is at once accompanied by failure of sight in the centre of the field of vision. Senile changes take place in the capillaries of the chorio capillaris just as they occur in the capillaries in other parts of the body, and changes in the macula due to retino-choroiditis, the result of old age, are not uncommon. Such

changes may be revealed by impairment of function for a considerable time before any pathological lesion can be detected with the ophthalmoscope. In the normal condition the pigment layer of the retina hides the choroid from view, and as long as that layer is undisturbed no ophthalmoscopic changes are visible. That explains why it is that an elderly person may complain of inability to read for a considerable time before any explanation of the failing sight is seen with the ophthalmoscope. The chorio capillaris supplying the macula may be seriously affected by senile changes, but not until the pigment epithelium is implicated does the macula lose its colour and delicate granular appearance. From this stage onwards the progress of the degeneration can be seen with the ophthalmoscope. Through a gap in the pigment epithelium the vessels of the choroid appear yellowish white, owing to thickening of their walls at the expense of their calibre, and when finally they become completely obliterated, they can be seen no longer, and the site of the macula is represented by a small white spot, all the vascular structures overlying the sclerotic having become atrophied."

CHAPTER XV

The Heart-beat

The parts concerned in the heart-beat.
(*a*) The genetic system.
(*b*) The muscular system.
Development of the heart.
The relation of the genetic to the muscular system.

The parts concerned in the heart-beat.—The circulation of the blood through both the systemic and pulmonic systems is maintained mainly by the force of the contraction of the ventricles. Other agents, as gravitation, the contraction of muscles and respiratory movements, help to a slight extent. The heart itself is, however, quite sufficient.

(*a*) *The genetic system.*—It has long been known that the heart contains within itself the means of maintaining its activity, so that when removed from the body it continues to contract in a normal manner so long as it is supplied with suitable nourishment. This fact indicates the presence of some structures endowed with the properties of causing the muscle to contract. The possession of this property has been traced to a structure which is peculiar to the heart, and which at present is inappropriately called the 'conducting' system. The investigation of the functions of this system has revealed certain forms of vital activity, which so far have not been recognized in any other structure in the body. It possesses the property of generating and discharging impulses which result in the contraction of the chambers of the heart. Hence the term 'genetic system' is employed in preference to the 'conducting system' (see Chapter XVIII.).

The recognition of this structure and its functions necessitates a profound alteration in our conceptions of the heart-beat. Nearly every observation, experimental and clinical, which deals with the movement of the heart, requires revision, for the interpretations were made at a time when the essential structures and their functions concerned in the regulation of the heart-beat were not recognized. Even yet our knowledge of the part these structures play is imperfect.

(*b*) *The muscular system.*—To obtain a knowledge of the manner of the heart-beat, inquiries must be made in different directions, such as a comparison with other muscular organs, the manner in which structures and functions are evolved, consideration of the different methods by which impulses are

conveyed to the muscle, the origin of impulses and other influences concerned in the contraction of the muscle.

Contraction is the chief function of the muscle cell, but it only occurs in response to an impulse from without itself. The variations in the strength of the contraction of a muscle depend upon the number of muscle cells taking part in the contraction, and upon the source of the impulses. There is a remarkable similarity in the manner in which the contractions of different muscles are regulated and controlled, although there is a wide difference in the structures which originate and convey the impulses to the muscles. This is shown in the resemblance between the contractions of the voluntary muscles and those of the heart muscle. Thus voluntary muscles contract in three different ways: first, by impulses from parts of the central nervous system; second, by impulses from other sources when it is quiescent, giving rise to that form of contraction called 'tone'; and third, by impulses which reach the muscle from other sources when it is severed from its nerve connexion, giving rise to that form called 'fibrillation'. These three forms are also found in the heart muscle. Impulses which cause contraction of the auricle and ventricles arise in the genetic system, starting normally at the s.-a. node. When the heart is not actively contracting, it still maintains a measure of contraction called 'tone'. When the influence of the genetic system is lost the muscle still contracts from other sources in that form of contraction called 'fibrillation'.

Development of the heart.—Notwithstanding these resemblances the different muscular organs show reactions which are peculiar to each. This is due to the difference in the structures which are concerned in regulating the muscle contraction.

The responses of organs, even when so much alike as the hollow muscular organs, are brought about by means which are peculiar to each organ. The peculiarity of each regulating mechanism is, to a great extent, due to the manner in which the organs were developed. The structures concerned in regulating the heart movements were developed along a line followed by no other muscular organ.

If we would understand aright the functional peculiarity of each of the structures concerned in regulating the heart-beat, we must go back to the development of the heart in order to find out how each of these structures came to be adapted to its present purpose.

At an early stage of its development the heart is a simple tube composed of muscle cells. These cells generate the impulses which cause contraction, and the impulses pass on with each contraction from cell to cell, so that at this stage muscle cells generate impulses, contract, and discharge impulses. These functions are so co-ordinated that the contraction of the individual cells is regulated and controlled in a manner which enables the organ to effect its purpose. The properties of impulse production and conduction *without control* are seen in muscle cells when taken from the primitive heart and grown in suitable nutrient fluid. There they develop into a thin sheet, and exhibit movements which are purposeless and ineffective.

In the course of development a stage is reached in which pouches are developed on the sides of the tube, which ultimately become the auricles and ventricles (Fig. 11). During this development the primitive cardiac tube loses some of its functions, while it becomes highly specialized for the performance of its other functions. The contractile function almost disappears and is taken up by the more recently developed pouches which become the auricles and ventricles, while the impulse production and conduction functions, and the functions of regulation and control are retained by the remains of the primitive cardiac tube. At one stage of development of the heart, as in the tortoise, the chief portion concerned in the production of impulses is the sinus venosus,

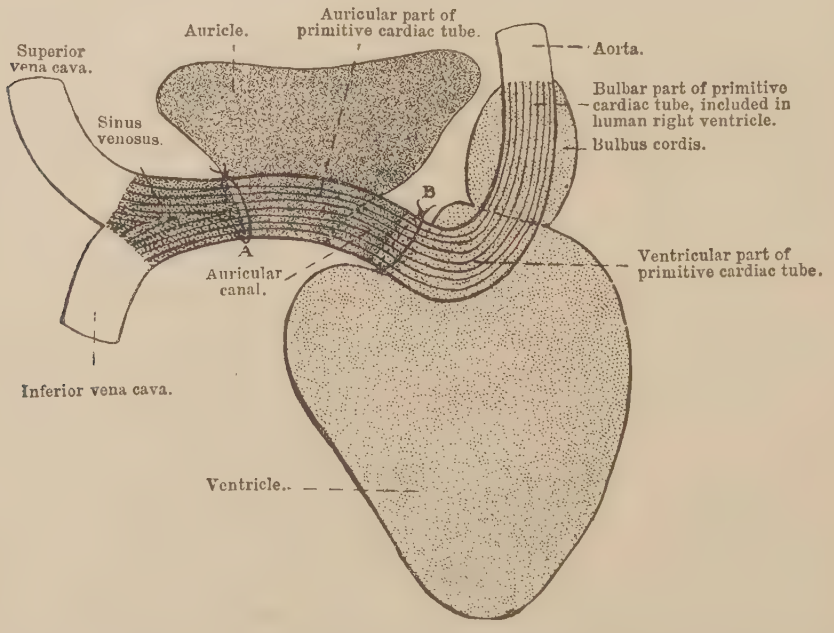


FIG. 11. Diagram of the primitive vertebrate heart, showing the development of the auricle and ventricle from the primitive cardiac tube. *A*, position of first Stannius ligature; *B*, position of second Stannius ligature. (Keith.)

which is also contractile. The primitive cardiac tube can be recognized in the canalis auricularis, which lies between the sinus and the ventricle, and the impulses from the sinus venosus pass along the canalis auricularis, giving rise to the contraction of the auricle and then, passing on, give rise to the contraction of the ventricle. Where the canalis lies between the auricle and the ventricle a portion of it is a ring of tissue with remarkable properties which controls the contraction of the ventricle, and which is evidently the progenitor of the auriculo-ventricular node. Some of its peculiarities will be dealt with when the a.-v. node is described. Under normal conditions it shows no individual peculiarity, being under the control of the sinus venosus.

At this stage of development the whole heart is normally under the control

of the sinus, but if the sinus is cut away the canalis auricularis takes up the function of impulse production and starts the contraction of the auricle, which is followed by the contraction of the ventricle. When the ventricle is cut away from the auricle the remains of the canalis auricularis take up the function of impulse production and stimulate the ventricle to contraction.

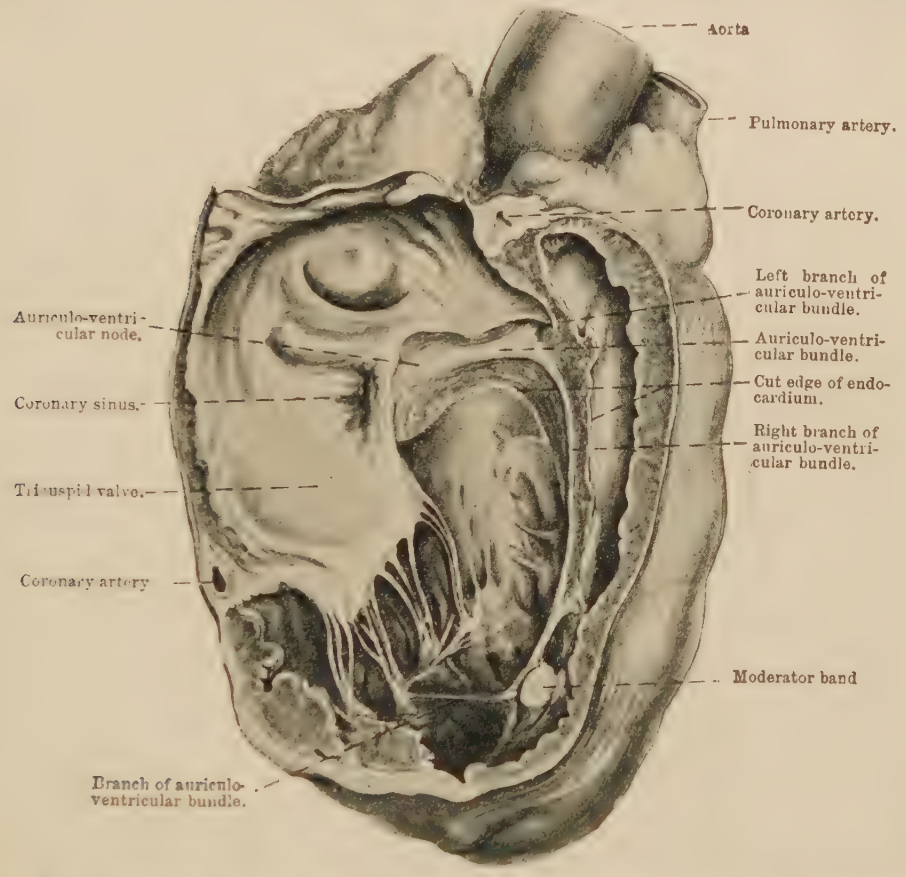


Fig. 12. Dissection showing the origin of the auriculo-ventricular bundle, and the course of the right branch in the right ventricle. The anterior and lateral walls of the right ventricle have been removed, as well as a portion of the tricuspid valve. A slight dissection has been made which exposes the bundle arising in the auriculo-ventricular node, an ill-defined structure in the right auricle, near the opening of the coronary sinus. The main stem runs from the node to the auriculo-ventricular septum, where it divides into two branches, the left branch going through to the left ventricle as shown in Fig. 13, the right branch going down in the muscle of the ventricle near the endocardium as a narrow rounded band. In the specimen from which the dissection was made, it could be traced to the moderator band, which is shown cut in the drawing. Here it divided into numerous fine branches, one of which is seen passing across the cavity as a fine thread.

These are the results which are found to follow after the first and second 'Stannius ligatures', the first ligature being between the sinus and the auricle,

and the second between the auricle and the ventricle (Fig. 11). In this interpretation of the results of these ligatures it will be seen that the contraction of both auricle and ventricle is dependent upon impulses from the remains of the original cardiac tube.

In the mammalian heart the distinctive characteristics of the tube have

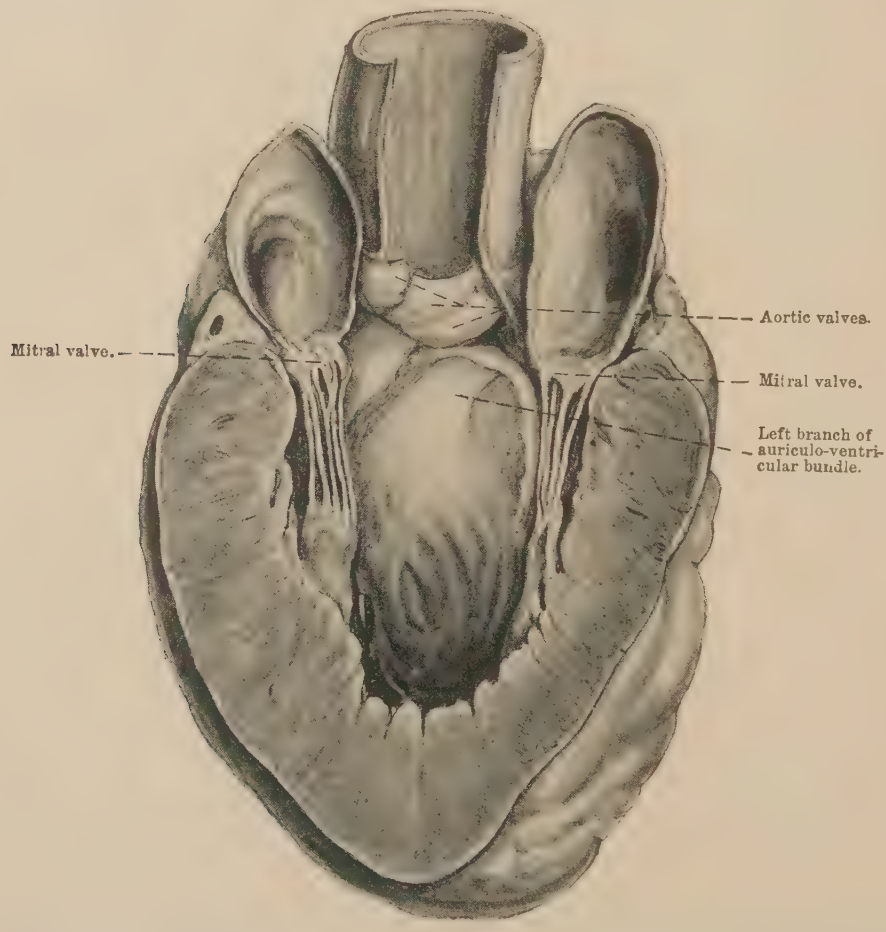


Fig. 13. Dissection showing the left branch of the auriculo-ventricular bundle. The posterior wall of the left ventricle has been slit open, the incision passing up through the left auricle and aorta. The cavity of the left ventricle is exposed, and a very slight dissection made to show the left branch of the bundle entering the ventricle immediately below the septal cusp of the aortic valve. The bundle widens out into a thin band lying immediately under the endocardium, and as it passes down the ventricle it sends branches into the muscle, until it can no longer be recognized as a separate and distinct structure.

disappeared, and there are left in its place structures which possess the function of impulse production and control. These structures are represented in the human heart in the sino-auricular node and the auriculo-ventricular node and bundle (Figs. 12 and 13). Although the cells of the bundle retain the

function of contraction to a slight extent, the effective contraction is taken up by the more recently developed muscle cells of the auricles and ventricles, which respond only to impulses conveyed by the genetic system.

From the consideration of the heart so far, it will be gathered that the auricular and ventricular muscles are entirely under the control of these regulating structures, so that the heart can have no purposive and effective functional activity apart from them.

This is a matter which has to be grasped if we are ever to understand not only how the normal heart performs its functions, but how its functional capacity becomes impaired as a result of disease.

To sum up—from the time that the heart was but a tube its functional activity was so controlled that it produced a response always purposive and effective. The influence which produced this result was retained by the primitive tube when the auricles and ventricles had developed. The different parts of the genetic system retain this influence in the human heart, and it is through its influence that the contractions of the auricles and ventricles are of that character which is typical of all normal muscular movements, viz. graded, purposive, and effective.

The significance of this will become apparent later, for there are conditions in which the chambers of the heart act in a manner that is purposeless and ineffective. When this happens it can safely be said that the disturbance arises from some derangement of the genetic system, that is, of the system which supplies the influence of control.

The relation of the genetic to the muscular system.—These two systems are associated together, and it is impossible to consider the one apart from the other. Just as it is not possible to understand the contraction of a voluntary muscle except in relation to its motor nerve, or the action of a motor nerve without its relation to the muscle, so it is not possible to understand a movement of the heart without a knowledge of the genetic system, or the genetic system without a knowledge of the muscle movements.

CHAPTER XVI

Anatomy of the Genetic and Muscular Systems

The structure of the sino-auricular node.
The structure of the auriculo-ventricular node.
The structure of the auriculo-ventricular bundle.
The muscle of the heart.
The blood supply of the heart.
The contraction of the cardiac chambers.

The structure of the sino-auricular node.—The sino-auricular node is a small mass of tissue composed of cells, striated and fusiform, with nuclei, embedded in connective tissue into which are distributed nerve fibres from the vagus and sympathetic. It is situated at the junction of the superior vena cava and auricle. There must, however, be other portions of the genetic system in the auricle which start the contraction, for when the s.-a. node is destroyed the heart-beat continues seemingly in the normal manner, though sometimes at a slower rate.

Experimental observations and the employment of the electro-cardiograph have shown that when the contraction arises elsewhere or from different parts of the s.-a. node, a different form of auricular wave is found in the electro-cardiogram. In the normal electro-cardiogram the auricular wave is upright, but when the impulses arise elsewhere the auricular wave becomes inverted.

This is well shown in Fig. 34, where the first few beats show an inverted auricular wave, but it gradually alters until at the end of the record the wave assumes the normal upright form. One would infer from such a record that in different parts of the auricle there are the remains of primitive cardiac tissue belonging to the conducting system.

The structure of the auriculo-ventricular node.—This is another mass of cells similar in constitution to those of the s.-a. node. It is situated in the wall of the right auricle, near the septum between the auricle and ventricle, close to the opening of the coronary sinus. The node is somewhat club-shaped and passes imperceptibly into the a.-v. bundle (Figs. 12 and 13).

Fibres from the auricle pass into this node, but whether these are the ordinary muscle fibres or whether they contain parts of the conducting system is not clear. This is of some importance, as the s.-a. node exerts an influence on the a.-v. node, which suggests some relation other than through the connexion with the auricular muscle.

Fibres from the vagus and sympathetic nerves are distributed to the a.-v. node.

Structure of the auriculo-ventricular bundle.—When the bundle leaves the node it passes over the septum and divides into two branches which pass respectively to the right and left ventricles. The bundle consists of a series of fibres made up of cells, larger than the ordinary muscle fibres and less markedly striated. They possess large nuclei enclosed in a special covering (Fig. 16, p. 101). The different fibres are connected with one another, so that each fibre of the bundle is continuous from its origin to its termination. In the ventricles the fibres break up into innumerable branches which interlace and form a complicated network. The cells of these branches have long been known as Purkinje fibres.

These branches ultimately terminate in the muscle fibres, but their manner of termination is not clear.

Nerve fibrils and nerve cells are abundant in both node and bundle, but it is not quite clear whether the vagus and sympathetic affect the bundle or its branches.

The muscle of the heart.—The muscle fibres of the auricle and ventricle are different from the muscle fibres of voluntary muscle and other involuntary muscles, in that they are striated and branch and anastomose, so that there is a continuity of the different fibres.

The blood supply of the heart.—The point of importance to note is the situation of the coronary arteries at their opening in the aorta. They are situated half an inch above the aortic valves. Under normal circumstances the supply of blood is greatly increased during effort, and in consequence the coronary arteries are very dilatable. As the aortic valves are frequently the seat of disease, the disease often extends to these openings, and may block them or render them so rigid that they do not dilate when the heart requires more blood during its activity.

There seems to be little intercommunication between the arteries of the heart. This fact is of importance, because when one artery is blocked the absence of a collateral blood supply favours the destruction of the muscle cells which it supplies—a fairly common condition in those who suffer from angina pectoris.

Such structures as the s.-a. node, a.-v. node, and the bundle have each a separate blood supply. This is also of significance, because when these arteries or capillaries are damaged so that the blood supply is diminished or cut off, the functions of these organs become impaired or lost, followed by a peculiar train of symptoms which are recognized as auricular fibrillation and flutter and heart-block.

The contraction of the cardiac chambers.—The manner in which these chambers contract has been a subject of many investigations and speculations. The peculiar manner in which the muscle fibres are arranged in the heart differs from that of any other organ. The tænia terminalis, the pectinate fibres of the auricle, and the auricular appendages, for example, are structures

that must have some peculiar part to play. Keith [1] has described how the regurgitation of blood into the superior vena cava is prevented by contraction of the broad band of muscle which sweeps over the roof of the auricle—the tænia terminalis. In its contraction this muscular band shuts off the vein from the auricular cavity. He considers that the circular fibres at the mouth of the veins are too weak to prevent regurgitation. As the tænia is attached at the orifice of the inferior vena cava, and is carried over the roof of the right auricle, it also aids in the closure of the inferior vena cava and the pulmonary veins. The pectinate fibres, he considers, not only assist in emptying the auricle, but pull up the ventricles by reason of their insertion into the auriculo-ventricular septum.

The contraction of the ventricle has also been a subject of discussion, and attempts have been made to explain its contraction from the consideration of the arrangement of the muscle fibres. Dissections by various people have resulted in a somewhat confusing description of the arrangement of the fibres. The central fibrous body, the pars membranacea, situated below the origin of the aorta, has been looked upon as really a tendon into which are inserted many muscle fibres. The muscle fibres at the apex have been described as constituting a whorl, in virtue of which the contraction of the fibres here constitutes a fixed point.

The muscle has also been considered as a syncytium in which the branched fibres link up the whole mass in such a way that the contraction takes place by means of this intercommunication. The discovery of the genetic system presents the subject in a new aspect. All muscle fibres are under the control of this system, and it must therefore play an important part in producing an effective contraction. The introduction of the electro-cardiograph has permitted the problem of contraction to be approached from another point of view, and elaborate descriptions of how the ventricle contracts, based upon the study of electro-cardiograms, have been put forward, but for reasons given (p. 426) it is doubtful if the electro-cardiograph can help in the solution of the problem.

I must confess that I have not been able to get a clear conception of the manner of contraction of either auricle or ventricle from the study of the different explanations that have been given. There is still such a great deal that is obscure, not only in regard to the manner in which the chambers contract, but to the manner in which the muscle fibres are stimulated and regulated in their contraction, and there is the further difficulty that the normal contraction takes place in a manner different from the experimental. This renders all experimental results suspect.

[1] Keith, A., 'The Evolution and Action of certain Muscular Structures of the Heart', *Lancet*, Feb. 27, 1904; also 'The Anatomy of the Valvular Mechanism round the Venous Orifices of the Right and Left Auricles', etc., *Proceedings of the Anatomical Society of Great Britain and Ireland*, November 1902.

CHAPTER XVII

The Nerve Supply of the Heart

The afferent system of nerves.
The sympathetic and vagus nerves.
The relation of the sympathetic and the vagus nerve to the heart.
Reciprocal innervation.
The denervated heart.
Stimulation of the vagus.
Stimulation of the sympathetic.

The afferent system of nerves.—The application of the principle of the reflex arc has shown the necessity for reconsidering many features of the heart-beat, and particularly the part played by the nerves. Hitherto attention has been directed almost entirely to the efferent fibres of the sympathetic and vagus, little consideration being given to the afferent fibres, and practically none to the other nerves with which the efferent fibres of the sympathetic and vagus are intimately connected—that is the afferent nerves from the various organs of the body.

Efferent nerves are stimulated only by impulses which they receive from other nerves. The sympathetic and vagus do not themselves originate impulses, nor are their efferent fibres directly stimulated by any of the organs of the body. The impulses which stimulate them are conveyed by the afferent fibres with which all the organs of the body are supplied (see Fig. 14).

While such a statement as this has been accepted in a general way its real significance has been missed. It has long been recognized that an increase in the influence of the sympathetic increases the rate of the heart, but it was not realized how the sympathetic itself came to be stimulated, nor how it caused the increased rate. Stimulation of a nerve or other organ is brought about by the increase of the number of impulses which affect the nerve or organ, and the question arises. Where are these impulses produced ? Such impulses can only arise from cellular activity. It can be shown that one great source of impulse production is the contraction of muscle cells, as in uterine contractions (see p. 326), when the impulses not only increase the heart-rate but cause a number of other reactions. A simple experiment demonstrates that the contraction of voluntary muscles can also produce such an increase in the number of impulses that the heart-rate is increased.

The mechanism by which the heart's rate is increased in response to effort

has been a problem which has hitherto baffled attempts at solution. The key to the solution is to be found in the following simple experiment. A continuous record by means of the sphygmograph was taken of the pulse of a man when lying on his back. After the rate had settled down he was asked to contract the muscles of the leg. When this was done the rate of the heart increased at once, and the increased rate continued for a short period after the muscles were relaxed. Fig. 15 shows this result. The signal at X shows the time the muscles began to contract, and the increase in rate begins at the second beat, the rate rising from 60 beats to 80 beats per minute. This is a fairly typical response, but it is not a constant one. Sometimes there is no response, sometimes it is delayed or affects only a few beats. Moreover, it is impossible to limit the muscular contractions to the muscles that move the foot, for the muscles of the arm and the movements of respiration are affected at the same time. Impulses from the brain can increase the heart's rate. These observations are recorded here to demonstrate that contraction of voluntary and involuntary muscles and mental activity influence the heart-rate, and these organs can only do so by generating impulses which play upon the heart through the sympathetic and vagus nerves.

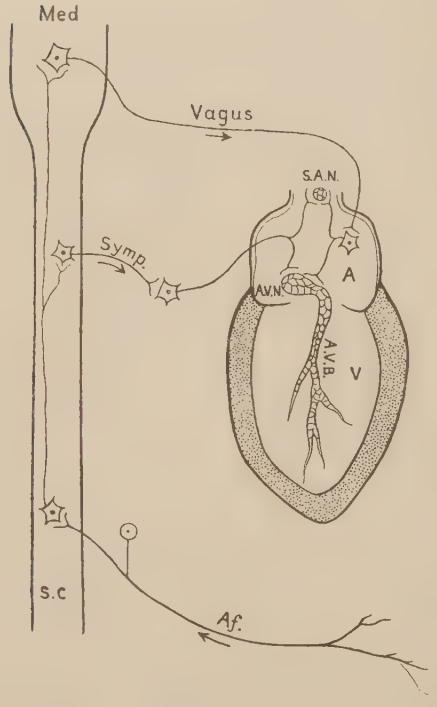


Fig. 14. Diagram to show the parts concerned in regulating the rate and rhythm of the heart. *Af* represents the afferent nerves from the different organs which convey impulses through the spinal cord (*S.C.*) and medulla (*Med.*) by neurones which stimulate the sympathetic and vagal nerves. These nerves are distributed in the heart to the sino-auricular node (*S.A.N.*) and the auriculo-ventricular node (*A.V.N.*). Impulses are conveyed from the sino-auricular node to the auricle which cause its contraction, and from the auriculo-ventricular node by the bundle (*A.V.B.*) to the ventricle (*V*).

Impulses can also be conveyed by the sympathetic and vagus nerves from the heart muscle itself. Such impulses stimulate the efferent fibres of these nerves which affect the heart-rate just like the impulses from the other organs of the body. There is no doubt that it is the afferent fibres of the vagus and sympathetic which convey impulses which produce pain and other reactions, as happens in the contraction of the uterus. This matter will be dealt with more fully in describing the nature of angina pectoris.

While it is usual to find evidences of an increased rate from impulses produced in the different organs (resulting from stimulation of the sympathetic)

from certain other organs, the impulses conveyed by the afferent nerves affecting the vagus nerve decrease the heart's rate. This is particularly the case in certain affections of the bowel, when impulses arising from it produce a slowing of the heart and syncope.

In a recent publication L. Grossi[1] has gathered a number of instances illustrating cardiac reflexes. From his description one can infer that every organ of the body is intimately related to the heart through the vagus and sympathetic nerves, in the manner shown in Fig. 14.

The sympathetic and vagus nerves.—The efferent fibres of these nerves to the heart have their receptors in the central nervous system, while their discharging ends, on which their peculiar function depends, are distributed mainly, if not entirely, to the sino-auricular and auriculo-ventricular nodes (Fig. 14). It is recognized that the stimulation of the sympathetic results in an increase in the rate and in the strength of the ventricular contraction, and is therefore spoken of as the 'augmentor' nerve of the heart. Stimulation of the vagus, on the other hand, produces a weakening of the heart-beat and a decrease in

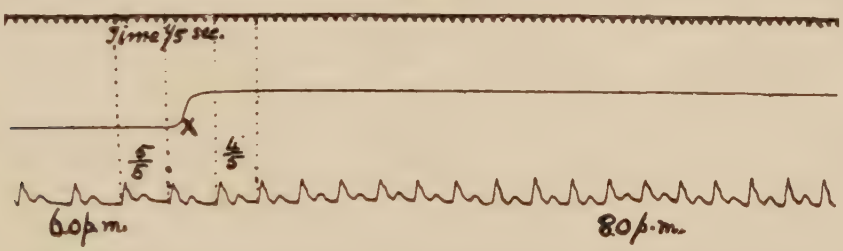


Fig. 15. Showing the increase in pulse-rate on contraction of the muscles of the leg. The signal X indicates the time of contraction. The rate increased from 60 beats per minute to 80, and began after the first beat (from five-fifths of a second to four-fifths per beat) and lasted for a few beats after the contraction had ceased.

its rate, and it is therefore spoken of as an 'inhibiting nerve of the heart'. From such results the conclusion has been drawn that these nerves acted directly upon the muscle cells of the heart. But this is one of the dangers of drawing conclusions from an end-result. No consideration has been given to the chain of structures which lay between the part to which a stimulus was applied (the cut nerve) and the contraction of the muscle. As we shall see later, the contraction of the muscle of the ventricle is entirely dependent upon impulses from the a.-v. node. If a large number of impulses are thrown out by the node, a strong contraction of the ventricle results, and if a few impulses are thrown out a smaller number of the fibres contract. As we know that these nerves do act upon this node it seems unnecessary to assume that they also act upon the muscle cells of the ventricle.

The relation of the sympathetic and the vagus nerve to the heart.—So far as the study of the functions of the different parts of the heart has gone, it would seem that the contractions of the chambers are entirely under the control

[1] Grossi, L., *Reflessi Cardiaci*, Geneva, 1923.

of the genetic system. The s.-a. node seems to possess the power of influencing not only the rate of the heart, but the manner in which the auricles and ventricles contract. It is therefore possible that it is only on the node that the efferent fibres of the sympathetic and vagus nerves act, when they modify the contraction of the heart when the rhythm is normal. When there is an abnormal rhythm, as in fibrillation or flutter of the auricle, they then act upon the a.-v. node, but as will be shown there is not that perfect regulation and control of the contraction of the ventricle which is exercised by the s.-a. node.

The afferent fibres of the sympathetic and vagus, on the other hand, are in direct communication with the muscle cells, for it is through the impulses thrown out by the muscle cells that the great complex of symptoms included in the term angina pectoris is produced.

Reciprocal innervation.—When two nerves having functions so distinctive and so opposed as the sympathetic and vagus supply the same organ, there is a curious arrangement by which they perform their functions. It is customary to speak of an acceleration of the heart-beat as being due to a stimulation of the sympathetic, and the slowing of the heart-beat has been due to the vagus.

When, for instance, the rate varies on account of the movements of respiration, it is said to be vagal in origin because it disappears when the vagus is cut. This is not a sufficient explanation, for the removal of one influence merely allows the other to be uncontrolled. It might as well be said that the slowing of the heart after effort is due to vagal influences, just as the increase in rate is due to the sympathetic influences. It is more reasonable to assume that when impulses increase sympathetic activity, they at the same time decrease the vagal activity, and *vice versa*.

It will be seen that this ' reciprocal innervation ' has practically the same result as that shown in the stimulation and inhibition which occurs when the hair of the sundew bends (see p. 52).

The denervated heart.—Some light is thrown upon the relation of the sympathetic and vagus nerves to the s.-a. node when the heart is completely freed from these nerves. In Starling's[1] experiments with the heart-lung preparation all the nerves to the heart were cut. By varying the arterial pressure the strength of the heart-beat could be made to vary within certain limits. But however varied the strength of the beat, the output remained practically constant *and the rate of the heart did not alter*. By increasing the inflow of venous blood, considerable dilatation of auricle and ventricle could take place, *with no variation in the rate of the heart.*

From these experiments it is seen that the s.-a. node is quite independent of and indifferent to the work of the auricles and ventricles, and that their distension has no direct effect upon the genetic system so far as the *rate* of the heart-beat is concerned. The fact, however, that the heart varied in the strength of the beat shows that there is within the heart itself a regulating influence, and this regulating influence can only be due to the genetic system.

It is not clear how this influence which regulates the strength of the beat

[1] Starling, E. H., *The Linacre Lecture on the Law of the Heart*, Figs. 2 and 3.

is produced. The factors concerned are chiefly the genetic system and heart muscle, but there is present a nervous system which may exercise a control on the activity of the genetic system similar to that of the sympathetic and vagus nerves upon the s.-a. node in regulating the rate of the heart.

Stimulation.—If the vagus nerve be cut, its influence upon the s.-a. node ceases, and the action of the sympathetic is unrestrained and the heart's rate increases. If the peripheral end of the cut nerve be stimulated the rate of the heart is at once reduced. From this it is inferred that the peculiar attribute of the vagus nerve is situated in its discharging fibrils (p. 69). This is further shown by the action of atropine. If given in sufficient quantity the vagus action ceases and the heart's rate is increased. If, then, the nerve is cut and its peripheral end stimulated, no effect is produced, for the reason that the atropine has acted by paralysing the peripheral fibres of the nerve.

If an intact vagus nerve be stimulated by digitalis, its effect is entirely lost if the nerve be cut. From this the conclusion is drawn that the *intact* nerve is stimulated only by impulses taken up by the fibrils at its receiving end (receptors). The increased activity of the nerve is therefore dependent upon its reception of an increased number of impulses. This increased activity of the vagus nerve can only occur in two ways: (1) by the receptors being assailed by an increased number of impulses; or (2) by an increased excitability of the receptors. The former method occurs when there is an increase of the number of impulses reaching it by the afferent nerves by which it is connected (as in the syncope which follows stimulation of the bowel).

The increased activity of the nerve due to the excitability of the receptors is seen in the effect produced by drugs or other agents which act upon the nerve. While atropine acts upon the peripheral fibrils, digitalis acts upon the receptors, increasing their excitability. When the excitability is increased they take up a great number of the impulses which are normally present.

The nerve, like all other organs, is subject to the law of fluctuation, so that the neurones of which a nerve is composed do not all act at one time. 'Stimulation' means an increased number of neurones brought into activity, and digitalis acts by doing this. Emetics act by increasing the excitability of the receptors of a greater number of the neurones of the nerves concerned in vomiting, so that impulses normally present give rise to vomiting. In many people digitalis acts as an emetic, and frequently at the time it produces vomiting it also produces heart-block. It is evident from this that the digitalis had rendered the receptors of both the vagal and other nerves concerned in vomiting highly excitable.

Stimulation of the sympathetic.—Stimulation of the sympathetic does not give such typical reactions as stimulation of the vagus, but there is no doubt that it is acted upon by drugs and the agents of disease in the same way as the vagus, as will be shown when we discuss the parts on which its effects are produced in describing the sino-auricular and auriculo-ventricular node.

CHAPTER XVIII

The Function of the Genetic System of the Heart

Introduction.
The generation of impulses.
The rhythmogenic function.
Conductivity.
Control.
The part played by the nerves in control.
The impulses which stimulate the contraction of the heart.
The law of ' all or nothing '.

Introduction.—A review of the history of the knowledge of the heart-beat during the last fifty years affords an illustration of the steps which favour and hamper progress in medicine. After the recognition of the relation of the nervous system to the muscular, the idea became prevalent that the nervous system originated and controlled all muscular movement in the higher animals. The heart was included in this idea, and ' motor heart nerves ' were spoken of, and also ' cardiac centres ' in the nervous system. Even when it was recognized that the heart could beat when removed from the body, the idea still held that it was dependent upon an inherent nervous system. Gaskell cast doubts upon this view. Romanes in his work on the medusa had described the muscle as conducting the impulses which caused the contraction. Gaskell applied this idea to the interpretation of certain of the reactions of the heart which he had obtained where he could positively state that no nerves were involved in the stimulation. Although Romanes' interpretation was afterwards found to be incorrect, in that the contraction of the muscle of the bell of the medusa is regulated and controlled by a nervous network, still Gaskell's observations on stimulating the canalis auricularis, which is a muscular structure, showed that there was something other than the nerve which controlled and regulated the heart-beat. He put forward the view of the myogenic origin of the heart-beat as a provisional hypothesis. In the last conversation I had with him he said that certain facts had come to light which made him doubt the validity of the hypothesis, and that there was some factor which he did not comprehend.

Since that time there has been revealed a series of structures the recognition of whose function has thrown a flood of light upon the heart-beat.

The generation of impulses.—It was unfortunate that Tawara,[1] when

[1] Tawara, S., *Das Reizleitungssystem des Saugethierherzens.* Jena, 1908.

he described the anatomy of the auriculo-ventricular node and bundle and its branches, in place of using an anatomical term employed one which is supposed to indicate the functions of the structures, 'Das Reizleitungssystem'—the 'stimulus-conduction system'. It has since then been taken for granted that the function of this system was mainly conductivity. Not only is this description unsatisfactory, for the system has other important functions, but it is misleading in that the system does not 'conduct' impulses in the sense which the term conveys.

As the term 'conducting system' is misleading, it is proposed to substitute the 'genetic system', for reasons that will appear later.

Had it not been taken for granted that the function was known, a much more thorough investigation into the functions of this system would undoubtedly have been undertaken, although it is questionable whether its function could have been discovered by the methods which guide investigation in the present day. It is a matter beyond experimental research, though experiment can and has contributed something to the knowledge of the subject. It requires clinical observations, and the use of methods and principles of a particular kind.

The need for a particular kind of inquiry is that the functions of this system, like its structure and its development, are different from those of any other structure of the body, so that there is no standard to act as a guide to the interpretation of its function.

It has been pointed out that in the primitive cell all the functions of highly-developed and specialized cells are to be found in a rudimentary state. When primitive cells perform their functions they also discharge a form of energy—the cell impulse (p. 49). This impulse is a peculiar and distinctive form of energy capable of stimulating other cells to activity. In the course of development certain cells of the body lose practically all their functions, and become specialized for the discharge of these impulses. This is particularly the case in the cells of some portions of the brain.

The rhythmogenic function.—The fact that the heart can beat without the intervention of nerves gave rise to the view that the heart muscle possessed a form of automatism which enabled it to beat of its own accord so long as it was supplied with suitable nourishment, and this action has been referred to as a 'rhythmogenic function'.

In the discussion of vital activities it was shown that living cells were never at rest, being engaged either in discharging energy or in renewing it (p. 60). It was also shown that where cells were gathered together to form an organ, the influence of one cell induced the discharge of energy in contiguous cells, so that an organ was never at any time at rest. It was also shown that cells exerted a control upon one another, and that this influence resulted in groups of cells discharging their energy at different times—sometimes many doing so at the same time. This group activity may be periodic, giving rise to a rhythmic activity. The sino-auricular node is one of those structures where this form of activity is highly developed, and it is to it that the rhythm of the

normal heart is due. ' Automatism ' is thus seen to be but a regulation of cell activity, and is fundamentally akin to all other forms.

Conductivity.—When Tawara described the ' Reizleitungssystem ' the whole problem of the conduction of impulses in the heart seemed on the point of being solved, and it appeared to be one of the easiest subjects to understand. The structures were looked upon as mere conductors of impulses, and having the same function as nerves. As physiologists and others had spent a great deal of time on the investigation of nerve conduction, conductivity was thought to be well understood. But when the principles of nerve conduction, so far as they were understood, were applied to the conduction of impulses through the genetic system, there were found such divergences that it became more and more manifest that in the sense in which the term is applied to the passage of an impulse along a nerve fibre, *the genetic system is not a conducting system.* The study of this system casts doubts on the accepted teaching of the conduction by nerves, and resulted in the explanation given on pp. 56 and 57. Looking at the matter broadly, one can see there were bound to be differences. The constitution of the individual cells, the structure of nerves and of the a.-v. bundle, and the manner of development, are widely different.

It has been shown that nerve cells become specialized for the purpose of conducting impulses. Such cells to a great extent lose most of their other functions, and the cells develop in such a manner that special parts are modified to carry on this function. From the cell body of the neurone there issue fibres and fibrils which are concerned in the reception of impulses, the conduction of impulses—it may be for short or long distances—and the discharge of the impulse by another set of fibrils. The cell body evidently does not take part in this function, presiding seemingly over nutrition. These facts are well recognized, and the impression has arisen that such structures as the genetic system act in the same way. But this is not so.

In primitive cells, as has been said, when the cell discharges its function it at the same time throws out impulses that affect neighbouring cells, and stimulates them to activity. This is what occurs in the primitive heart, where the muscle cells when they contract throw out impulses which stimulate the neighbouring cells.

Gaskell referred to the canalis auricularis as being the remains of the primitive cardiac tube, and described the auricles and ventricles as having developed on the sides of this tube and gradually taken on the function of contraction, while the tube gradually lost this function of contraction, while maintaining the function of regulating and controlling the movements of the auricle and the ventricle.

The origin and structure of the genetic system is thus widely different from that of the nervous system, and the function of conduction of impulse to the effector organ is carried on in quite a different manner. In the cells of the auriculo-ventricular bundle (Fig. 16) there are no specially developed parts like the fibrils and long fibres of the neurone. Each is a small compact body which, in addition to the function of discharging impulses, retains

the function of contraction. Recent observations by Ishihari and Nomura[1] show that branches of the auriculo-ventricular bundle contract at the same rate and rhythm as the ventricle itself. Even if taken out of the heart and placed in a suitable fluid, the bundle will continue to beat for a long time (see p. 61).

This has a very important significance, for it shows that this system in the conduction of impulses does so after the original and primitive manner, that is to say, it discharges an impulse at the same time that it is contracting, thus differing from the conduction in a neurone as usually described.

It also means that each part of the system is capable of taking up the function of the s.-a. node, that is, of generating and discharging impulses which result in a contraction of the auricle or ventricle.

When this aspect of the functions of this system is appreciated, it will be found to afford an explanation of the seemingly confused rates and rhythms which at present baffle all attempts at a classification on a rational basis.

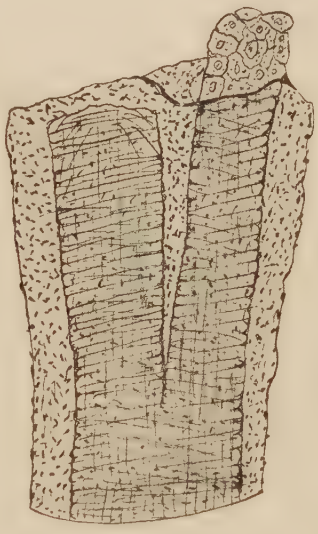


Fig. 16. 'False tendon', showing bundles of Purkinje conducting tissue inside an elastic cellular sheath. The bundle divides, and one branch projects from its sheath and there shows its cellular structure. (×120.) (Waterston.)

Control.—The possession by the cells of the genetic system of the function of impulse production and contraction and the variations in the excitability of the cells are not sufficient to enable the heart to do its work. In addition, there is required the function of control, which has been described (p. 59). This function is of two kinds, one exercised by the genetic system on the muscle of the auricles and ventricles, and one exercised by the nerves on the genetic system.

Control by the genetic system.—It has already been shown (p. 61) that in the normal activity of every organ the structures that discharge the impulses exercise also a function by which they regulate and control the action of the organ so that its contraction is carried out in a particular manner, being purposive and effective, and graded to meet the circumstances of the moment. This function is exercised by the genetic system, so that it regulates and controls the movements of the auricles and ventricles. When it is lost the muscle fibres of these chambers contract from impulses from other sources in an ineffective and purposeless manner. This function of control, again, is possessed by every part of the genetic system. It is exercised over the muscle when the muscle is seemingly at rest. When at rest the muscle exhibits a slight

[1] Ishihari, M., and Nomura, S., 'On the Contraction of the Branches and Terminal Ramifications of the Auriculo-Ventricular Bundle in the Heart', *Communication to the XI. International Physiological Congress*, 1923. See also *Heart*, vol. x, 1923, p. 399.

degree of contraction called 'tone', the result of impulses conveyed to it by the nerve in the case of the voluntary muscle, and by the genetic system in the case of the heart muscle. While the muscle is at rest the function of control is exercised, so that the muscle fibres are prevented from responding to impulses from other sources. When this function is lost the muscle fibres respond to impulses from other sources, and pass into fibrillation.

While each part of the genetic system possesses all these different properties they have them in an unequal degree. Under normal conditions the s.-a. node is the most excitable portion, so that it discharges impulses before the other portions have become sufficiently excitable to discharge them independently. In this respect all the other parts of the genetic system are dependent upon the impulses which originate in the s.-a. node. The function of control is also possessed unequally, and the sino-auricular node exercises a control over the remainder of the genetic system. When this control is lost, as when the s.-a. node ceases to exercise its function, the a.-v. node behaves in a different manner, in that it becomes much more susceptible to influences or agents from the outside (as shown in auricular fibrillation and flutter).

Control by the nerves.—The control by which the heart's movements are regulated to meet the requirements of the body is dependent on the connexion of the s.-a. node with every organ through the vagus and sympathetic nerves (Fig. 14). The s.-a. node is so susceptible to the influences of these nerves that it readily accommodates the heart's activity to the varying activity of the other organs. When the heart is removed from the body and suitably fed, it still contracts, but there is no longer the power of regulating its activity. When the s.-a. node is thrown out of activity and the contraction of the ventricle is dependent on the a.-v. node or bundle, this fine adjustment of the heart to the varying circumstances of the body is lost (see p. 106).

The impulses which stimulate the contraction of the heart.—Although there is still much that is obscure about the heart-beat we can now get clearer ideas of some aspects of the contraction. The s.-a. node in its development passes through a stage when its cells are endowed with the function of contraction as well as that of impulse discharge. The former function becomes attenuated while the latter has been retained. It is not the impulses which this node produces which stimulate the muscle cells of the auricle and ventricle. The impulses from the cells of the s.-a. node stimulate only the cells with which they are directly in contact, and each cell as it receives this impulse contracts and discharges an impulse to affect in turn neighbouring cells, so that the passage of impulses follows, not the method which is assumed to occur in the highly developed neurone, but that primitive method found in the hairs of the sundew and in the primitive cardiac tube. The impulses, therefore, which cause the contraction of the chambers are the impulses from the cells of the genetic system immediately in contact with the muscle cells, and not those which were generated by the s.-a. node.

The law of 'all or nothing'.—When we reflect that the stimulus for

contraction arises in and reaches the ventricle by means of a specialized structure acting in a particular manner, and that this structure enters the ventricle at a particular place and breaks up into innumerable branches so that muscle fibres are stimulated to contract in a particular sequence, and compare this contraction with that where an abnormal stimulus like electricity reaches the ventricle, not in the normal manner nor at the normal place, it is manifest the result cannot be the same.

Bowditch, on applying an electrical stimulus to any part of the ventricle, found that the whole heart contracted whether a strong stimulus or a weak one was applied, so long as it was effective. From this he deduced what is called the law of ' all or nothing ', that when the ventricle contracted it did so with all the energy of which it was possessed. This law has been implicitly accepted as physiological, whereas it is merely an experimental and unnatural result.

In our investigations we find grounds for believing that no organ carries on its work according to the law of ' all or nothing '. The functions of organs are so regulated and controlled that their response is always graded to suit the circumstances of the moment, a few cells participating in the discharge of function at one time and more cells at other times, *i.e.* according to the law of fluctuation, and not to the law of ' all or nothing '.

The importance of this distinction cannot be overestimated, for so long as the belief is held that the heart carries on its work according to the law of ' all or nothing ', it will be impossible to understand how the heart modifies its activity to respond to effort, while the variations in the rate and rhythm of the heart in health and disease will remain inexplicable.

CHAPTER XIX

Classification of the Variations in Rate and Rhythm of the Heart

The advantages of a classification.
Variations due to increased excitability of different parts of the genetic system.
Variations due to decreased excitability of different parts of the genetic system.
Variations due to the cessation of activity of parts of the genetic system.
Table and diagram of the abnormal rhythms.

The advantages of a classification.—The classification of phenomena according to their affinity in nature permits not only a better appreciation of the phenomena, but serves as a guide for further investigation. The test of the validity of a classification is to be found in satisfying these requirements. In Chapter XIII. a classification of symptoms was given, based on their relation to the vital processes concerned in their production, in which all symptoms could be referred to influences which increased or decreased the activity of living structures or interfered with the influences which regulated and controlled their activity.

The application of this classification to heart irregularities affords an opportunity for testing its usefulness. In due course evidences will be forthcoming showing that the classification holds good. It will be found that all irregularities are due to an increase or decrease of activity of the different parts of the genetic system, or an interference with the function of control.

Hitherto classifications have been based on superficial resemblances, and have failed to bring out the relation of one form of irregularity to others. In consequence it has not been possible to recognize the nature of individual irregularities, or to see clearly what line to follow to acquire this kind of knowledge, while the essential purpose of the recognition of the irregularity for the practice of medicine—its prognostic significance—could only in rare cases be obtained, and that by methods already described of watching for long years individual patients, while the principle of the investigation of prognosis, described in Chapter III., could not be applied.

Though the validity of a classification be recognized, it is not possible to know at first how to use it effectively or to foresee where it may lead. The classification which resulted from the atomic theory is an illustration, so in the classification of the irregularities given here it is not possible fully to comprehend its significance, but experience is showing a way in which it can

be applied, and in this and the following chapter some of the results are given.

Variations due to increased excitability of different parts of the genetic system.—The increase and decrease in rate of the normally acting heart are due to an increase or decrease in the excitability of the s.-a. node. The feature of these variations in rate and rhythm is that they are gradual in both the increase and decrease.

As any part of the conducting system may become more excitable than the s.-a. node, the ventricles then respond to impulses from such excitable part. The impulses then anticipate those from the s.-a. node, and the contraction of the ventricle occurs earlier than it otherwise would. Such contractions may occur singly or in groups, and may start in the different parts of the genetic system. The single contractions are extremely common, and are recognized as the ' extra-systole '.

While usually the disturbance leads only to the production of a single beat, occasionally a series of beats may follow one another at a rapid rate, and may continue for brief or long periods. Such periods are spoken of as attacks of ' paroxysmal tachycardia '.

As these beats break in upon the dominant rhythm, *i.e.* the rhythm which originates in the s.-a. node, the paroxysm begins suddenly. On the cessation of the paroxysm the dominant rhythm resumes control, and as it is set at a much slower rate, the termination of these attacks is always sudden.

It will thus be seen that all forms of increased rate of the heart are due to an increase in the excitability of limited portions of the genetic system. The normal rate of the heart varies according to the excitability of the s.-a. node, and the causes are the agents that increase or decrease its excitability. The abnormal rates are due to an increased excitability of other limited portions of the genetic system.

Variations due to decreased excitability of different parts of the genetic system.—Just as all parts of the genetic system are liable to increased excitability, so are they liable to decreased excitability. In the normally acting heart, when the pulse-rate falls from a higher rate the fall is due to a decrease in the excitability of the s.-a. node.

Other parts of the genetic system may be rendered less excitable, and cause a delay in the passage of impulses from the s.-a. node to the auricle and ventricle, or prevent impulses reaching the auricle and ventricle. Such disturbances are only recognizable by the late appearance of the ventricular systole, or by the failure of the ventricle or auricle to respond to the impulses from the s.-a. node. Such conditions result in that form of decreased ventricular rate which is termed heart-block. When one part of the genetic system is too slow in performing its function or fails to do so, the part nearer the ventricle may take up the function.

Variations due to the cessation of activity of parts of the genetic system.—When the disease process is such as to destroy or put out of action one part of the system the other parts carry on their functions, but each part, although

possessed of the faculty of independent activity, possesses individual peculiarities. There are three parts where the independent activity can be studied: (1) the normal starting place in the s.-a. node; (2) the a.-v. node; and (3) the bundle. When the s.-a. node ceases to function or is destroyed, the a.-v. node takes up its independent activity, so that the ventricle continues to contract. When the a.-v. node ceases to function or is destroyed the bundle takes up its independent activity and starts the contraction of the ventricles.

These three are not alike in their independent activity. The s.-a. node possesses in a high degree that function of control by which the rate of the heart and the strength of the beat are varied to meet the requirements of the body. This is brought about by its connexion with the vagal and sympathetic nerves. It also regulates and controls the a.-v. node and bundle, and when it ceases to function, not only is the auricle uncontrolled (fibrillation), but the a.-v. node is also freed from control and exhibits peculiar features.

Under normal circumstances the a.-v. node is subject to the control of the s.-a. node. It has not that function of regulation and control so highly developed as the s.-a. node. It is supplied by the sympathetic and vagus, but does not respond to them with the delicate purposive efficiency of the s.-a. node. The consequence is that the ventricle reacts in a manner which differs remarkably from its behaviour when the s.-a. node is in control. This is well seen in certain patients with auricular flutter. At rest the rate may be about 50. A slight bodily effort that would increase the normal heart-rate 5 or 10 beats increases the ventricular rate to 150, at which rate it fails to respond, either by increase or decrease, to the varying activity of the body (Figs. 7-9). The same sort of ill-regulated excitation follows effort in many hearts with auricular fibrillation. There may be an inordinate increase in the rate with small imperfect beats, and no adjustment to meet the varying bodily activity (Fig. 107).

When, as in complete heart-block, the independent a.-v. bundle governs the ventricle, there is a total absence of that function which regulates the ventricle to meet the requirements of the body.

Another reason for the difference in the functional activity of these three structures lies in their surroundings. The s.-a. node is practically uninfluenced except by the regulating influences of the sympathetic and vagus nerves. When this node ceases to function, the auricle passes into fibrillation and flutter, but the a.-v. node, lying in the wall of the auricle, is exposed to the influences of the impulses discharged by the muscle cells of the auricle as well as to those of the sympathetic and vagus nerves.

When the a.-v. node is destroyed the bundle takes up the function of starting the contraction of the ventricle, and is not exposed to those influences which excite the two nodes.

Table and diagram of abnormal rhythms.—In the following table there are arranged three columns, one showing the modified process, another column showing the reactions which these modified processes produce, and the third column giving illustrations drawn from the heart.

Modified Process.	*Reaction.*	*Illustrations.*
Number of impulses increased.	Increased activity.	Extra-systoles and tachycardias.
„ „ „ decreased.	Decreased „	Heart-block.
Loss of control.	Disorderly „	Fibrillation of the auricles.

The diagram Fig. 17 is constructed to show how all the abnormal variations of the heart-beat can be classified according to the structures and vital processes concerned.

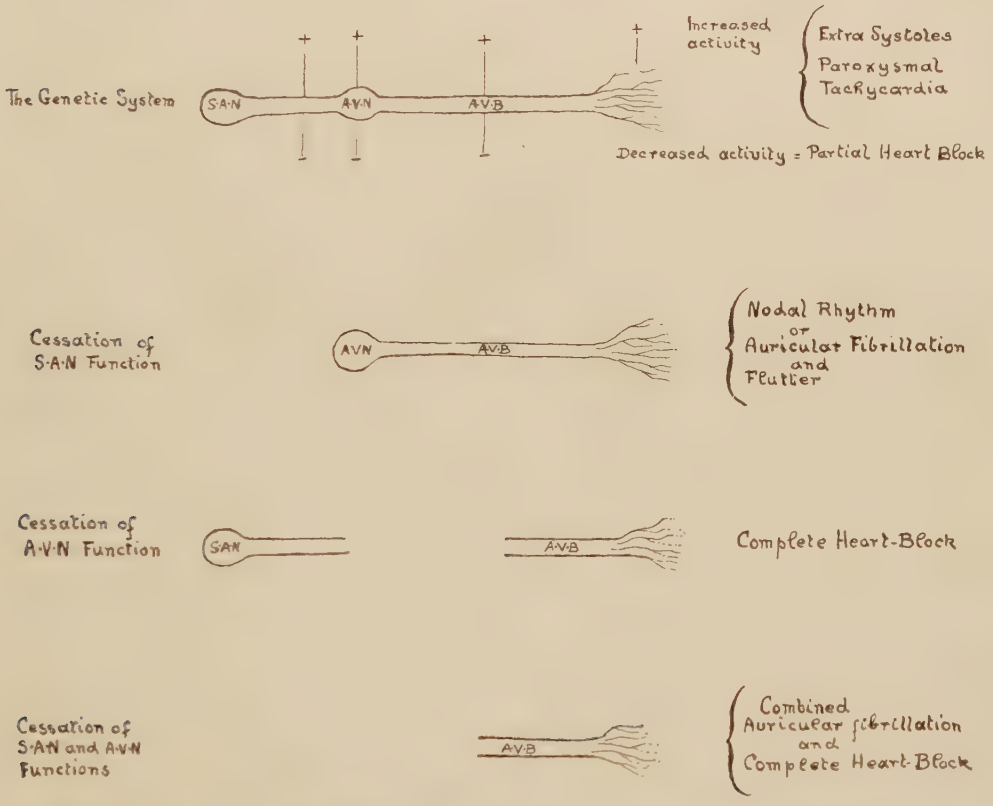


Fig. 17. Diagram showing the structures and processes concerned in producing the various abnormal rhythms. An increased activity (shown by +) of different parts of the genetic system gives rise to the different kinds of extra-systole, and certain forms of paroxysmal tachycardia. A decreased activity (shown by −) gives rise to partial heart-block. When the s.-a. node ceases to exercise its influence (shown by the absence of the s.-a. node) the a.-v. node starts the rhythm of the ventricle (nodal rhythm), and the auricle passes into flutter or fibrillation. When the a.-v. node ceases to function (shown by the absence of the a.-v. node) the a.-v. bundle starts the rhythm of the ventricle (heart-block). When both nodes cease to function (shown by the absence of the s.-a. and a.-v. nodes) the auricles fibrillate or flutter and the bundle starts the rhythm of the ventricle.

CHAPTER XX

The Place of Instrumental Methods

The use of graphic and electro-cardiographic records.
The training of the senses.

The use of graphic and electro-cardiographic records.—In the descriptions given of various phenomena of the heart the purpose has been always kept in view of helping the doctor in the examination of his patients. Stress is therefore laid upon the detection of the phenomena by the unaided senses. Where instrumental methods are employed preference is given to those which will record the signs that can be perceived by the senses. Graphic records, therefore, are employed in recording the movements of the radial pulse, of the apex beat, and of the jugular veins, because these are the movements which the doctor can see and feel, and must learn to recognize and to interpret.

Graphic records are employed in preference to those obtained by the electro-cardiograph, because they are far more informative. We know the forces that produce the various waves which are shown by a graphic record, but it is not known what the agent is that produces the electro-cardiogram (see pp. 424, 425). All that can be said from an electro-cardiogram is that the auricle or the ventricle contracted, and in some instances these chambers contracted in peculiar ways, but what these ways are no one can tell. Information can thus be obtained of the relation between the movements of the auricles and the ventricles, a kind of information obtained with equal certainty by graphic methods. It is only in rare instances the electro-cardiograph reveals movements which it is not possible to observe in other ways. The electro-cardiograph is in some respects an accurate instrument and of use in experiment, but its limitations in clinical medicine are great.

It is well to remember that there are matters essential to intelligent practice, such as the capacity of the heart for its work, which no instrument can reveal.

The training of the senses.—This method of examination by the training of the senses is gradually going out of fashion. It must be obvious to any reasonable man that if discoveries in medicine are to be made available in practice the doctor must be instructed so that he can apply them in his daily work. The other day a learned physician and teacher in a large medical school was called to see a patient with some cardiac disorder. After examining the patient he gravely announced that it would be necessary for the patient to be brought to his consulting-room to have an electro-cardiogram taken, for

he suspected the presence of auricular fibrillation, and if this suspicion were confirmed he would then prescribe digitalis !

This is not an exceptional incident, rather it is typical of modern practice, and shows that the trend of modern medicine is to render the doctor more and more unfitted for his work.

If this is read by a young teacher who has not become fossilized he should recognize that it is incumbent upon him, if he would do his duty by his students, to teach them to recognize the essential symptoms of disease by the unaided senses, and he should start by training himself. There is no sign of importance exhibited by the heart which cannot be recognized by the unaided senses. It behoves the teacher, then, to make himself familiar with the essential phenomena, and in place of relying upon the polygraph and electro-cardiograph for the detection of abnormal movements he should train his index finger to feel the radial pulse, his ear to detect the different sounds of the heart with all their variations, and to perceive with his eyes the movements in the veins of the neck, and to correlate the information obtained by these methods with that which an instrument may supply. In this way he is brought into intimate contact with the patient and with the phenomena of disease.

To attain this kind of knowledge requires intense application and a great deal of hard work, with the employment of every available mechanical device, till the physician is able to do without them. He will soon see to what it leads, and discover the value of the knowledge to be beyond anything that he had suspected. Moreover, he will find that he has taken a great step forward in appreciating the value of the signs which reveal the heart's functional capacity, which after all is the object of his examination.

It is for these reasons that in the descriptions to be given I use graphic records freely and electro-graphic sparingly in the endeavour to describe the signs by which the variations in the heart's rhythm can be detected by the unaided senses.

CHAPTER XXI

The Normal Rhythm

The liability of the s.-a. node to disturbance.
The normal rate of the heart.
The increased pulse-rate.
The significance of the increase in the pulse-rate.
The rate of the heart as a test of cardiac efficiency.
Diminished frequency with the normal rhythm.
Differentiation.
Differentiation by the unaided senses.
Prognosis.

The liability of the s.-a. node to disturbance.—The structures concerned in the variation of the heart-rate and rhythm are shown in Fig. 18. The genetic system is a structure with all the attributes of an organ. It is composed of cells which have specific functions ; it is supplied with blood, and it is under the influence of nerves. When it has discharged its function the cells take some time to recover. The period can be hastened or delayed as in other cells, and it is therefore exposed to a great variety of influences which result in the increase or decrease of its activity.

Besides being exposed to such functional disturbances it is, like all other organs, liable to disease, and therefore to impairment of function. As yet we know so little of these diseases and their effects that it has not been recognized where to look for them.

Having a regulating function, evidences of its activity will be found in the structure it regulates, that is, in the movements of the auricles and ventricles. When we find a heart disturbed in any of the three ways which result in a symptom, *i.e.* increased, decreased, or disorderly activity (pp. 105-106), we can conclude that the source of the disturbance is to be found in the s.-a. node, or in the parts which are allied to it in its function of producing impulses.

The normal rate of the heart.—The rate of the heart varies considerably according to age, sex, and individual peculiarities. At birth the rate is usually from 130 to 140 per minute. With advancing years the rate decreases gradually. Between the ages of 9 and 10 it falls to about 90, while at 20 it is about 74, and at 30 between 66 and 76. After this the rate does not vary much till over 50, when it begins to increase gradually in frequency.

At all ages there are considerable variations, and many healthy people may exhibit rates in adult life as high as 100 beats per minute, or under 50.

The increased pulse-rate.—In the normally acting heart variations in the pulse-rate are dependent upon variations in the activity of the cells of the s.-a. node. This variation is dependent upon the rate of the recovery of the cells after they discharge their function. The cells of this node act in the manner already described, where the cell, after discharging its function, takes

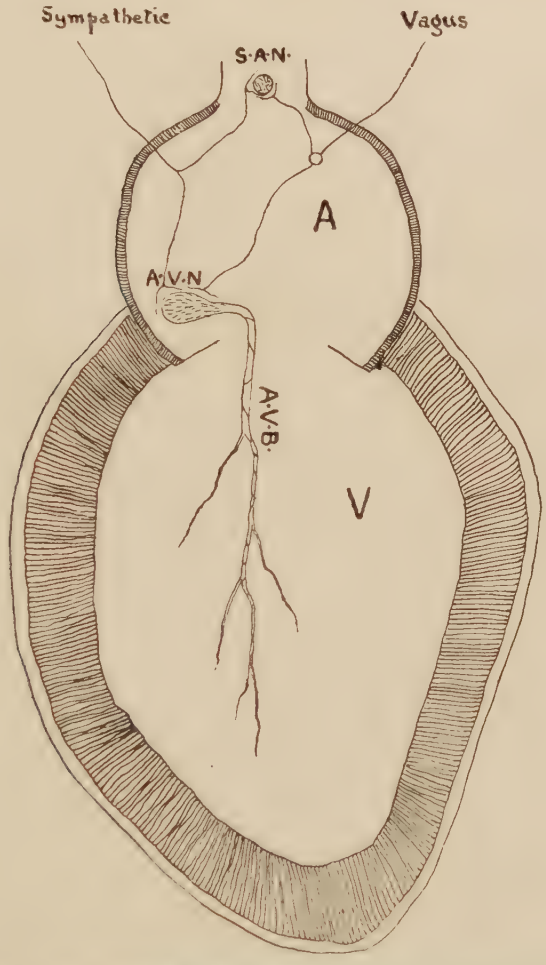


FIG. 18. Diagram of the structures concerned in the movements of the heart. *V* is the ventricle and *A* the auricle. *S.A.N.* is the sino-auricular node, *A.V.N.* the auriculo-ventricular node, and *A.V.B.* the auriculo-ventricular bundle. The sympathetic and vagus are represented as being distributed to the two nodes, as it is on them they act in regulating the rate and rhythm of the heart.

some time before its store of energy is renewed. The rate of renewal can be influenced by a number of agents. The most common of these agents are the sympathetic and vagus nerves. The manner in which these nerves influence the rate of the pulse in bodily effort has already been described (p. 94). It

has been shown that the increased rate comes from stimulation of the sympathetic nerves or diminished activity of the vagus. The increase in rate is far from being constant in different individuals. The amount of effort which will raise the pulse-rate to 120 beats per minute in one healthy person, may raise it to 200 beats and over in another. Similar differences occur on milder efforts.

In the same way great differences exist in the time required for the pulse-rate to return to the normal. In a trained, vigorous man the return may be slow and delayed. In untrained men the normal rate may return in a much less time. That is to say, that in perfectly healthy individuals an increased rate in response to effort, and the time which it takes for the rate to return to normal, vary with the individual, the reason evidently being that the excitability of the cells of the s.-a. node is not a constant factor amongst normal individuals.

It has been pointed out that the agents of disease may disturb the functions of every organ in the body. One of the organs most susceptible to the agents of disease is the s.-a. node, an increased rate of the pulse being a feature in many forms of ill-health. This is due to an increase in the excitability of the s.-a. node.

The most typical increase of rate takes place in febrile complaints. This has been looked upon as a result of the rise in temperature. While heat is one of the agents that hasten the recovery of the cell after it has discharged its energy, it is not the only agent concerned in increasing the rate in febrile complaints. This can be shown by observing an individual in different febrile illnesses. In one illness with a temperature of 101° the pulse-rate may be about 90 per minute. In another febrile attack with the same temperature, or even a lower temperature, the rate may be 120 per minute. From such observations it can be inferred that the different attacks were due to different agents, and that the difference of agents accounts for the difference in pulse-rate.

The significance of the increase in the pulse-rate.—These descriptions of the variations in the pulse-rate may seem so obvious that it is not necessary to dwell upon them, but if correctly understood they have an important and hitherto unsuspected bearing upon the practice of medicine. In the first place, it is to be observed that these variations in rate are due to variations in the excitability of the s.-a. node, so that when the *rate* of the pulse is counted it only gives information relative to the s.-a. node. The reason that this is emphasized is because the variations in rate have been taken as a measure of the heart's functional capacity, a matter with which variations of rate have nothing to do. In the same way in various forms of ill-health, as in febrile complaints, the rate of the pulse is taken as a measure of the gravity of the patient's condition. This again is a great mistake. It is true that in serious affections the rate may be greatly increased, but it is a mistake to take the increase of rate as an estimate of gravity. When a doctor intelligently feels the pulse, it is not so much the rate which gives him the information he wants, as the character of the pulse. A patient with puerperal fever may have a

temperature of 104°, with a full firm pulse, at a rate of 120 per minute. The patient's face may be flushed, and the patient quite intelligent. Such a condition carries no indication of gravity, although one may not be sure of complications that may arise. On the other hand, another patient may have a temperature of 102°, with a small feeble pulse at a rate of 110. The face may be pale or grey, and the patient dull and apathetic. These are indications of great gravity, the gravity being recognized not by the rate of the pulse but by its shabby character, and the associated signs.

There are individuals who are manifestly ill in whom the increased pulse-rate is the only objective indication of ill-health, except, it may be, laboured breathing on moderate effort. Many people take this increase of rate as an evidence of a heart affection, and they will even diagnose it as a myocarditis. The impression is fairly common that the myocardium has something to do with the rate of the pulse. There is no doubt that in acute infections of the heart the rate is increased—so it is also in acute infections of any other structure of the body. Even in acute endocarditis, the endocarditis has no more relation to the increased heart-rate than has acute inflammation of any other organ. That is to say, the increased heart-rate is produced in acute affections of the heart by the agents of disease acting on the s.-a. node, as they do in any other inflamed organ.

While acute myocardial diseases are associated with an increased rate, the vast majority of myocardial affections, even when so advanced as to imperil life, cause no increase in rate. Furthermore, the response to effort in such conditions does not produce a greater increase in rate than it does in healthy hearts.

When we find a patient with a persistent increase of the heart-beat with the normal rhythm we should carefully scrutinize every part of the body for local disease, and the condition should not be attributed to an affection of the heart without other evidences than the increased heart-rate.

OBSERVATION 1.—In the early years of the war I was consulted by an officer who had been wounded in the abdomen by shrapnel. He had lain in hospital for some time, and the wounds had gradually healed, and he seemed to be doing well, but, according to the doctors, 'his heart gave way'. The reason for this was the occurrence and persistence of an increase in the rate of the pulse, 120-130 beats per minute. He was kept on his back for three months, and all sorts of remedies were tried. When I examined his heart I could find nothing amiss save the increased rate. Examining the abdomen, I found a number of keloid scars, with a good deal of tenderness on pressure of the tissues around the scars. I pointed out that the keloid and the tenderness indicated a microbic infection, and this was probably the cause of the increased heart-rate. I kept in touch with him for eighteen months—for a long period there was no change, but after fifteen months the keloid disappeared, and there was no longer any tenderness around the scars, and the pulse-rate gradually became normal, and he was able to return to his regiment feeling fit and well.

The purpose of this illustration is to show that an increased excitability of the s.-a. node is not indicative of a heart affection. It is true a microbic infection of the heart may produce toxins which raise the excitability of the s.-a. node, just as toxins from the abdominal wounds did in the case just mentioned.

The rate of the heart as a test of cardiac efficiency.—Another reason for insisting upon this point of view is the practice of using the increased rate of the heart as a measure of the heart's efficiency. If it be realized that the heart's efficiency is dependent upon the muscle and not upon the s.-a. node, it will be seen that a measure is used for a purpose for which it is unsuited. One of the most profitless lines of investigation has been those numerous and elaborate attempts to discover the condition of the heart's efficiency by various tests in which bodily effort is employed, and the heart-rate taken as a standard (see p. 28). If one reflects, it will be seen that while in some instances the increased rate may induce exhaustion of the heart muscle, the increased rate is not the result of exhaustion. The variability in the excitability of the s.-a. node has no relation to the heart muscle—in other words, while the movements of the ventricles are dependent on the s.-a. node, the s.-a. node is independent of the ventricles.

Diminished frequency with the normal rhythm.—This only occurs when all the chambers of the heart participate in the slow action. The demonstration of the character of the slowing is best shown by comparing the jugular pulse with the radial (Fig. 19) or apex beat (Fig. 20), where the auricle is seen to beat at the same rate as the ventricle. There are a number of people in the enjoyment of perfect health whose pulse beats regularly about 50 per minute. In some of these a rise of temperature of one or two degrees may actually make the pulse beat slower. There are a number of conditions which tend to decrease the rate of the pulse, as exposure to cold, increased arterial pressure. In pregnancy the pulse may also be occasionally slow. It is found not infrequently in patients with jaundice, and with typhoid fever.

I have seen two cases with pulse-rates of this kind, in one 36 per minute, in the other sometimes lower than 30 per minute. The latter was an acrobat, 40 years of age, and he had been himself aware of the slow heart-rate for twenty years. It would usually fall to its lowest during the night, and he would sometimes wonder if it would not stop altogether, the pauses lasting so long. During the day the slow periods varied with periods of increased rate but usually irregular action, like that shown in Fig. 19. Colonel Deane made a careful study of this man during his work and at rest, and reports that immediately after violent exertion the rate would be 140, falling in two minutes to 64 on stopping the exertion. Tracings of the jugular pulse (Fig. 19) and electro-cardiograms showed that all the chambers of the heart participated equally in the slow action. The former case was a clergyman, 54 years of age, who only complained of feeling tired after a hard day's work.

Differentiation.—As the doctor is usually guided by the pulse, it will be more practical to differentiate the forms of slow pulse-rate. In order to differentiate between the different forms of slow pulse-rate, it is necessary to make observation of the movements of the various chambers of the heart. If this is done, it will be found that the cases of diminished frequency of the pulse can be divided into four classes: (1) those where all the chambers of the heart participate in the slow action (normal rhythm) (Fig. 19); (2) where

the slow pulse-rate is due to a missed beat, the ventricle having contracted, but the resulting pulse-wave being too feeble to reach the wrist (Figs. 62 and 63); (3) where the excitability of the a.-v. node is decreased (as in certain cases of auricular fibrillation and flutter) (Fig. 137); (4) where the impulses after the auricle has contracted fail to reach the ventricle (Figs. 176, 177) (Heart-block, Chapter XL.).

It is the first of these that is dealt with here.

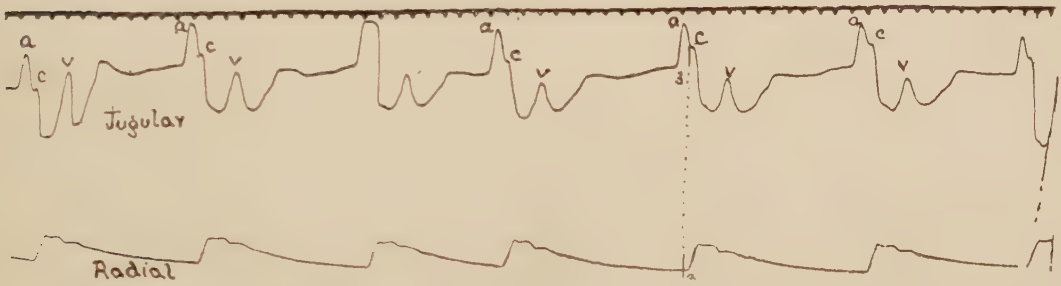


FIG. 19. Infrequent pulse from a healthy acrobat. Rate 26-30 per minute. The jugular pulse shows that all the chambers of the heart participate in the infrequent action.

Differentiation by the unaided senses.—There is usually little difficulty in differentiating different forms of slow pulse by means of records, graphic or electrographic. With training the doctor can differentiate the various forms by means of the unaided senses. By careful palpation of the pulse he may detect the small beats which might be overlooked by an untrained or super-

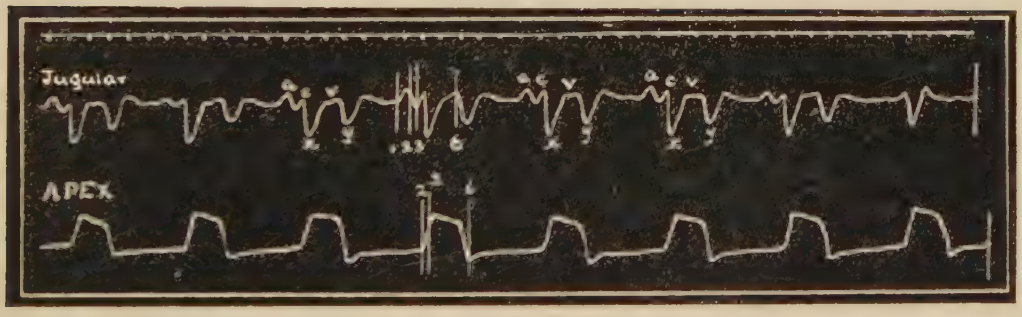


FIG. 20. Simultaneous tracings of the jugular pulse and apex beat, showing the participation of auricle and ventricles in true bradycardia. Rate 50 per minute.

ficial examiner. In the same way careful auscultation of the heart may detect feeble beats which do not send out enough blood to be felt at the pulse. The examination of the jugular pulse is of the greatest value, for we may observe in the slow pulse of a normally acting heart the auricle beating at the same rate as the carotid but immediately preceding it. In heart-block there usually is present a greater number of jugular pulse-beats than

of carotid. In the slow pulse of auricular fibrillation there usually is a jugular pulse at the same rate and synchronous with the carotid pulse.

Prognosis.—I have watched healthy men for many years leading active lives with a pulse under 50. Two of them I have watched for over forty years. They are now in good health at 70 and 74 years of age.

Where some agent causes the slow rate—jaundice—typhoid fever—the prognosis does not depend on the slow rate, but on the condition producing the slow rate. Within certain limits, as in typhoid, the slow rate is a favourable sign.

CHAPTER XXII

Disturbances of the S.-A. Node

The youthful type of irregularity.
Periodic variation in rhythm.
Standstill of the whole heart.
Other sinus irregularities.
Palpitation.
Prognosis of the disturbances of the s.-a. node.

Variations in the rate and rhythm of the whole heart are due to disturbances of the s.-a. node or the structures allied to it which start the heart-beat. There are several kinds of disturbance, but only a few have been clearly differentiated, and none of them studied with that particularity which will

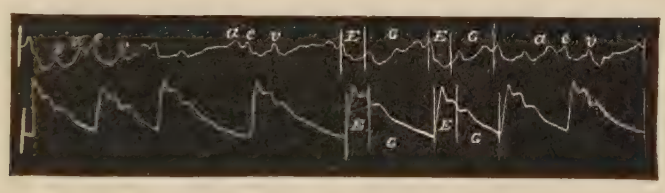


Fig. 21. Simultaneous tracings of the jugular and radial pulses, showing the agreement in rhythm of the right auricle and ventricle (waves *a* and *v*) with the radial pulse, in the respiratory form of irregularity. The irregularity is seen to be due to variations in the length of the diastolic period (spaces *G*).

enable us to understand how they differ one from the other. In this chapter I deal with a few of the more common forms.

The youthful type of irregularity.—The disturbances of the s.-a. node are represented by variations in the rate and rhythm of the heart. Such variations can be produced by the influence of the sympathetic and vagus nerves upon the node. The most striking of these variations is that form of irregularity found so frequently in the young.

This irregularity is easily recognized. To the finger the pulse-rate may be continually changing, but the beats are always fair and about equal strength. Graphic records show that all the chambers of the heart participate in the irregularity (Fig. 21). On auscultation the sounds are heard clear and distinct, and the interval between the first and second sounds is constant. By the ear a varying difference in the diastolic period can be made out more easily than by feeling the pulse. If the patient be made to

breathe slowly and deeply, the ear can detect with remarkable precision the relation of the slow and rapid actions of the heart to expiration and inspiration (Fig. 23).

From the description given of the afferent nervous system, it can be inferred with a fair certainty that the source of this variation is to be found in impulses thrown out by the muscles which are engaged in the movements of respiration, or from some other peripheral source. Such impulses conveyed into the central nervous system periodically affect the sympathetic and vagus. The time during which the effect on these nerves lasts may be longer than

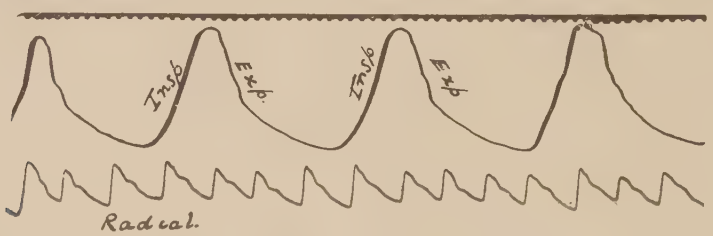


FIG. 22. Youthful type of irregularity showing respiratory variations—diminished rate during expiration and an increased rate during inspiration—more marked with deep breathing. (See Fig. 23.)

the period of inspiration, and in consequence there may not be a close relation between the variations in the pulse-rate and the movements of respiration until the latter become much slower.

When organs like the s.-a. node are supplied by two nerves having opposite effects, it is not possible to tell the part each plays in producing an effect,

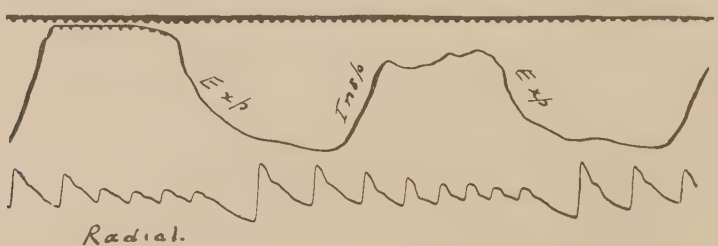


FIG. 23. Continuation of Fig. 22, when the patient breathed deeply and slowly. The increase in rate during inspiration, and decrease during expiration, are well marked.

such as this form of irregularity. It is, therefore, not correct to say this irregularity is vagal in origin because it disappears when the vagal influence is lost.

I have called this form of irregularity the ' youthful type ' because it is of frequent occurrence in the young, and it tends to become rare after 20 years of age, though some adults may show it late in life.

As a rule, if the breathing is held, either in inspiration or in expiration, this variation in rate disappears, but it occasionally persists for reasons which do not at present admit of explanation.

Periodic variation in rhythm.—There are instances where a similar irregularity occurs which is independent of the respiratory movements. Figs. 24-28 are tracings from a case of this sort, and they are given to show that although at first sight the irregularity seemed to be associated with the movements of respiration, yet a few simple experiments showed that it was independent

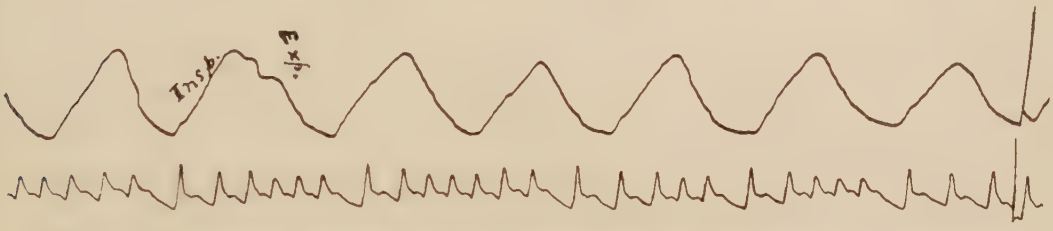


FIG. 24. Simultaneous tracings of the movements of respiration and the radial pulse, showing that the irregularity of the pulse has no relation to respiratory movements.

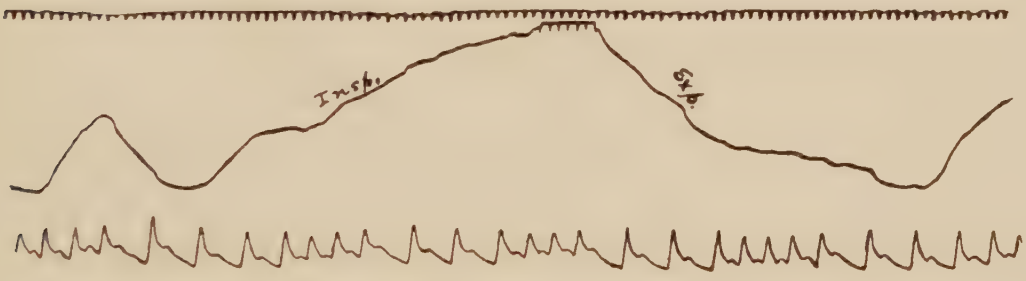


FIG. 25. Shows the persistence of the irregularity, with slow, full respiratory movements.

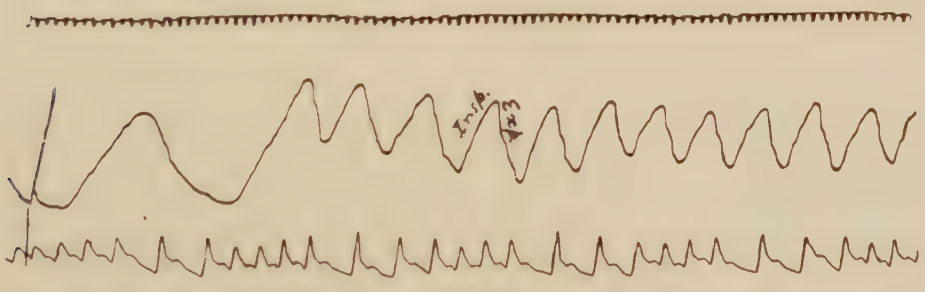


FIG. 26. Shows the persistence of the irregularity when the respiratory movements are accelerated.

of the respiratory movement. In Fig. 24, which is a tracing of the pulse with the movements during normal breathing, the pulse variations do not occur at the same time as the respiratory movements. Fig. 25 shows slow, deep inspirations and expirations. The same form of irregularity is seen to persist. In Fig. 26 the respiratory movements are increased from 15 per

minute to 30, but still the irregularity continues. In Fig. 27 the breath is held for a considerable time, and the irregularity of the pulse does not seem to be affected. In Fig. 28 is a tracing of the jugular pulse showing that all the chambers of the heart participated in the contraction, just as it does in the ordinary form of youthful irregularity.

These tracings were taken from a boy, aged 13, who was said to have had rheumatic fever three years before and was in bed for eight weeks, and was supposed to have had some slight attack since. He had fainted occasionally in church.

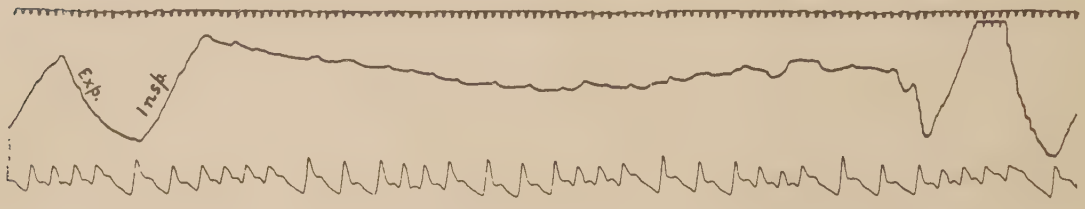


FIG. 27. Shows the persistence of the irregularity when the breath is held in inspiration.

On examination there was nothing found to be wrong except this irregularity. He was advised to lead the life of a healthy boy, and to play games at school. Six months later the irregularity was still present, respiratory in time, but when the breath was held the irregularity continued, but not so marked. Two years later the irregularity had completely disappeared, and the boy was leading an active life without any discomfort.

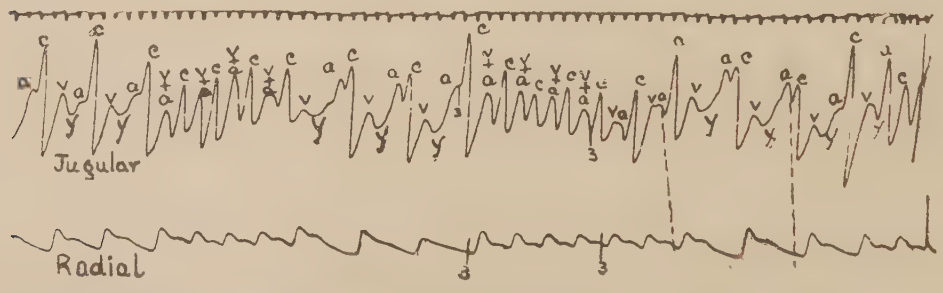


FIG. 28. Shows an irregular pulse of the youthful type. During the period of the infrequent rate, the *a* and *v* waves in the jugular pulse are separated. During the more rapid rate, the shortening of the diastolic periods causes the *a* and *v* waves to fall together.

Standstill of the whole heart.—Sometimes we come across individuals in perfect health in whom there are long pauses in the pulse, evidently due to the temporary suspension of the activity of the s.-a. node, as shown in Fig. 29. I have watched several individuals for ten or fifteen years, who exhibited this peculiarity at frequent intervals, and they never showed any cardiac impairment. On rare occasions digitalis produces a similar kind of standstill (Fig. 237, Observation 67).

There are rare cases in which a period of standstill is so long that the

patient loses consciousness. A typical case of this sort has been recorded by Laslett.[1]

Other sinus irregularities.—In old age there are varieties of irregular action of the heart which we must attribute to the effects of those degenerative changes so commonly associated with old age. As yet it is not possible to correlate the variations of rate with morbid conditions. A fairly common type of irregularity is short runs of rapid beats. The rhythm may be irregular

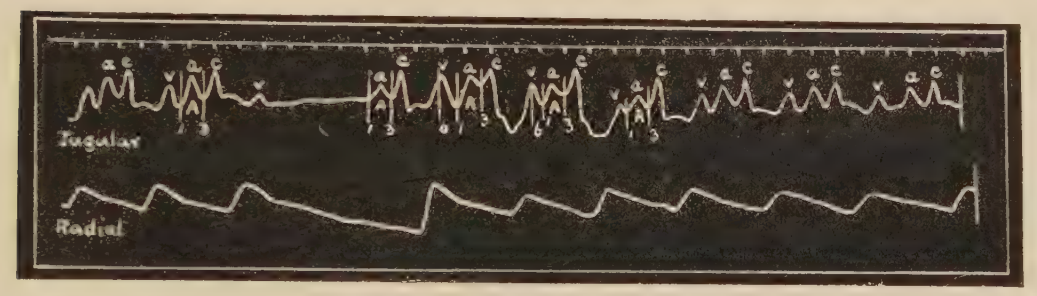


FIG. 29. Occasional slowing of the whole heart due to inhibitory nerve influences on the sinus. The *a-c* interval (space *A*) is not affected by the variations in rate.

during the rapid periods, or regular with long pauses. Fig. 30 is a typical instance from a man, 73 years of age, who had exhibited for many years a blood-pressure of over 250 mm. Hg. He led a fairly active life till the age of 79, when he died.

Palpitation.—A great increase in the rate of the pulse frequently arises from an undue excitability of the s.-a. node. It is not easy to tell whether

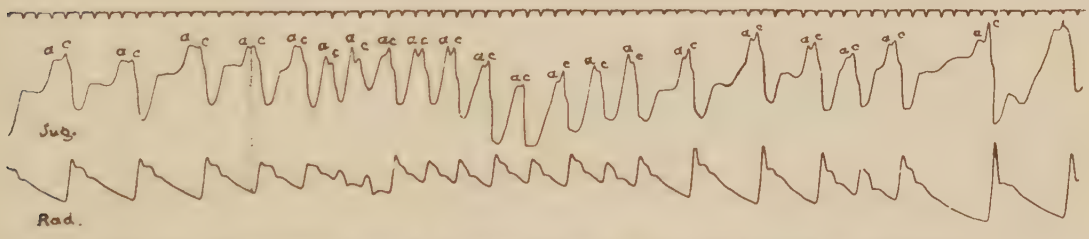


FIG. 30. Simultaneous records of the radial and jugular pulse showing an irregularity originating in the s.-a. node, probably due to senile changes.

the increase in rate is due to an increased excitability of the cells of the node from conditions affecting the node directly, or through increased sympathetic influence, or a decreased vagal influence. These increases of rate may occur at short periods and give rise to what is commonly known as attacks of 'palpitation', where the rate is greatly increased (Figs. 31, 33). The characteristic of this form of increased heart-rate is that though the attacks may

[1] Laslett, E. E., 'Syncopal Attacks associated with Prolonged Arrest of the Whole Heart', *Quarterly Journal of Medicine*, vol. ii. p. 347.

start somewhat abruptly they pass off gradually. The patient is frequently made conscious of the sudden increase in the heart-rate by a fluttering sort of sensation in the chest, but other people may not be conscious of its presence. It is often accompanied by a sense of exhaustion, and even by a sense of fear, as of impending dissolution, while in some people it causes not the slightest sensation, nor does it interfere with their bodily efforts.

These differences in the sensations are due probably to two causes, the one where the rapid heart-beat generates in the heart muscle a great increase in the impulses conveyed by the afferent fibres—the sympathetic or vagus

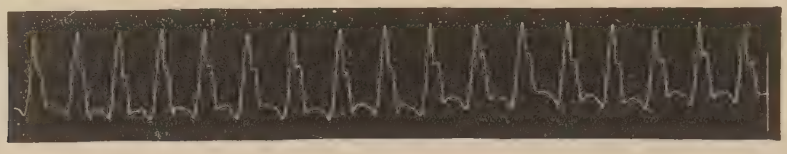


Fig. 31. During an attack of palpitation. Rate 105.

Fig. 32. Tracing of the normal pulse of the patient from which Fig. 31 was obtained. Rate 64.

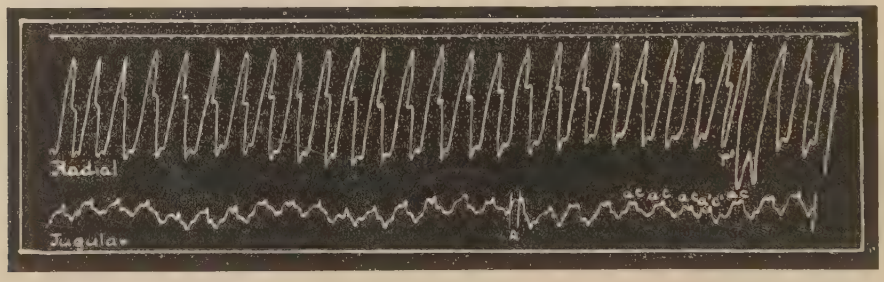


Fig. 33. Simultaneous tracings of the radial and jugular pulses. The rhythm is normal, the rate 164 per minute. There is an auricular extra-systole (a' and c' in the jugular tracing and r' in the radial).

nerves—producing reactions in the central nervous system, the other where the output from the heart is so diminished as to impair the cerebral functions.

The term palpitation is used in this book to describe the increased rate of the pulse which results from those disturbances of the s.-a. node, as distinct from the increased rate which results from an increased excitability of some other part of the genetic system, for which the term ' paroxysmal tachycardia ' is reserved.

The patient's account of his sensations will frequently serve to differentiate the two forms when the attack is not present—the passing off of the attack in palpitation is gradual, in paroxysmal tachycardia it is sudden.

Prognosis of the disturbances of the s.-a. node.—If we apply the principle which I have endeavoured to establish, that the prognostic significance of a symptom depends upon whether the function of the structure, whose disturbance gives rise to the symptom, is necessary to the maintenance of life, we get a guide to the prognosis.

It has already been pointed out that the essential matter in the prognosis of heart affections is the capacity of the muscle to do its work. All that the s.-a. node has to do with the muscle is to regulate its action. However disorderly its activity may be, it can only impair the functional efficiency of the muscle by a very greatly increased rate, or a very slow rate, and as these are of extreme rarity the ordinary evidences of disturbance of the s.-a. node are of no grave prognostic significance. The complete suspension of activity of the s.-a. node will be discussed when dealing with auricular fibrillation.

The notion was practically universally held at one time that the irregularity which I have called the youthful type was an evidence of some affection of the heart so serious as to require careful treatment, while the individual was considered unfit for occupations or games that required effort. I have seen large numbers of such persons rejected for life insurance, for the services, and made to submit to elaborate forms of treatment. If one cares to read the literature on this subject before I directed attention to this form of irregularity, such scant references as are to be found are accompanied with grave forebodings, while in practice the results were as I have just described.

As I have already mentioned, I have made a large number of observations upon this type of pulse, and watched many individuals who showed it from childhood up to adult life, and found it so common in healthy people that it can be considered a physiological manifestation.

But I did more than seek for its prognostic significance; I endeavoured to use it in a more positive sense, by seeing if its occurrence precluded the presence of other serious affections of the heart. In febrile conditions this irregularity tends to disappear, and with the subsidence of the fever tends to return. In cases where the heart might be affected, as in rheumatic fever, I observed that the appearance of this irregularity was an indication that the heart had escaped damage, even although a systolic murmur may have developed during the illness. The after history of the patients proved this to be true, and I was puzzled for a long time to find out the reasons. With the appreciation of the principle which I have laid down, one can understand the absence of any grave prognostic significance in that the disturbance of the s.-a. node does not embarrass the muscle in its work. The reason for its being of good omen in post-febrile cases was not at first quite clear, but it would appear to be as follows. It has already been pointed out that the s.-a. node is very sensitive to the toxins of microbic infection. The source of these toxins may be in any organ, and by stimulating the s.-a. node cause an increase of the heart-rate and abolish the youthful type of irregularity. If, then, in rheumatic fever the infection in the heart persists, its presence will be shown, as in all other infections, by a

persistent increase in the rate of the heart, while, on the other hand, the appearance of this irregularity will have the same indication as the fall of the temperature, showing that there is no active disease present.

While such is the reasoning I employ to explain prognosis of this kind, and I have never yet known any instance where it has failed, yet it is quite possible that there may be disease of a progressive kind in which there is no stimulation of the s.-a. node, and in which the youthful type of irregularity may be present. I have not, however, come across such an instance, although several people have called my attention to cases where progressive disease was associated with this type of irregularity. But on careful analysis of their records I am not convinced that the irregularity was the same. There was, indeed, occasional slowing of the pulse, but there was no indication that the variations in the pulse-rate were related to the movements of respiration. It seemed to me that the slowing was of a different kind, such as in Figs. 24-28. This is a subject which those with a suitable opportunity might reinvestigate.

While I thus explain my point of view, and the reasons for its acceptance, I recognize that while my own experience permits me to state that this youthful irregularity is physiological, it has not been sufficiently extensive to enable me to dogmatize about the presence of this arrhythmia being an evidence that the heart is free from acute infection.

CHAPTER XXIII

The Independence of the Different Parts of the Genetic System

The peculiar constitution of the genetic system enables each part to start a contraction of auricle or ventricle. There are occasions when parts of the system precede the s.-a. node in originating impulses. This is illustrated in Fig. 34, which shows that some part of the genetic system other than the s.-a. node started the contraction. In the normal electro-cardiogram the wave due to the auricle is upright. Lewis has shown that when the impulse arises from some part of the genetic system in the auricle other than the s.-a. node, the shape of the auricular wave alters, sometimes becoming inverted. In this record (Fig. 34) there is shown a series of auricular waves (P).

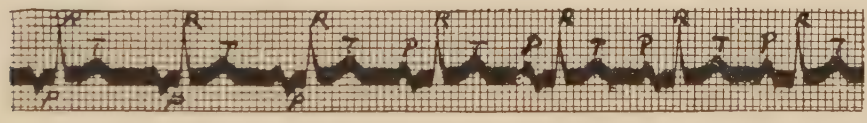


Fig. 34. Electro-cardiogram of a rhythm starting in the genetic tissue of the auricle and passing to the sino-auricular node. This is shown by the inversion of the P wave at the beginning of the record, and the gradual change of the wave to the upright at the end of the tracing, where the record is normal. (Cohn.)

At the beginning they are inverted, but gradually they alter until they become upright. This indicates that in the first instance the impulses had arisen in the conducting system remote from the s.-a. node, but gradually the starting-place had approached the s.-a. node until this node took up the rhythm of the heart.

The difference in the shape of the P wave indicates the different parts at which the auricular contraction began, so that the fibres in their contraction followed a different sequence than in the normal contraction.

The auriculo-ventricular node may become more excitable than the s.-a. node and anticipate or precede the impulses of the s.-a. node. This is shown in Fig. 35. Here the P wave due to the auricle precedes the R wave due to the ventricle in the first few beats, the auricle occupying the normal position, but gradually the interval between P-R lessens, then P and R become synchronous, and then the R wave precedes the P wave. As the ventricular wave R is dependent upon the a.-v. node, we recognize that in this instance the a.-v. node has taken up the starting of the ventricular rhythm.

A similar state of affairs is seen in Fig. 36, where the jugular pulse shows a curious variation in the length of the *a-c* interval. In some instances the *a* and *c* occur almost simultaneously, and seeing that the carotid pulse occurs some little time after the beginning of a ventricular systole, the auricular and ventricular contractions must have occurred together, and the ventricular systole was therefore due to impulses originating in the a.-v. node. It will

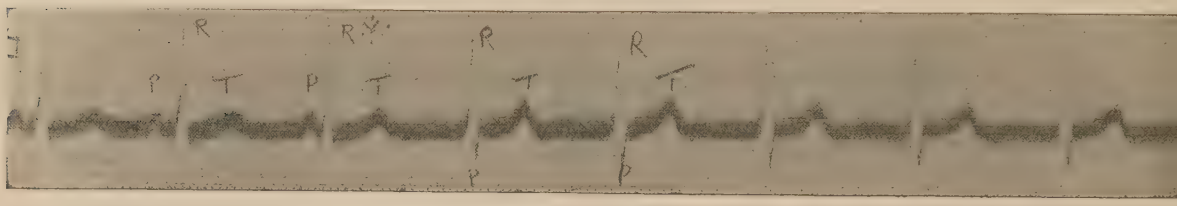


FIG. 35. Electro-cardiogram showing an altered action of the heart after the first two beats. The summit *P* is due to the auricular contraction, and the summits *R* and *T* are due to the ventricle. The third ventricular beat is independent of the auricular contraction. After this, auricle and ventricle contract together (probably a true nodal rhythm).

be seen from the radial pulse that the ventricular contractions were full and complete.

The activity of the a.-v. node, independent of the s.-a. node, occurs sometimes when the action of the s.-a. node is delayed. In some instances the s.-a. node becomes susceptible to digitalis, so that its activity is depressed, with the result that the heart stands still for a short period, and when it starts it may be due to impulses from the a.-v. node, as in Fig. 37, where there is no auricular wave preceding the carotid pulse in the first beat after the long

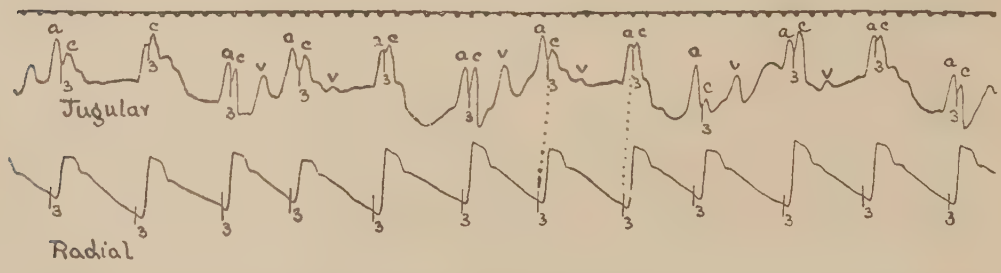


FIG. 36. The jugular pulse shows variations in duration of the *a-c* interval. When the interval is short, the auricle and ventricle contract simultaneously, due to an increased excitability of the a.-v. node, probably of the same nature as Fig. 35.

pause. The same thing happens occasionally in partial heart-block. Here the a.-v. node may not recover in time to respond to the impulse from the auricle, so that a ventricular beat drops out, as in Fig. 38. Before the next auricular beat is due the node has recovered and started a contraction of its own. This is seen in Fig. 38, where the wave *a'* is much larger than the other *a* waves because at that time the auricles and ventricles were contracting

together, and as the auricle could not empty its contents into the ventricle they were forced back into the jugular vein, giving rise to the larger wave. The impulse for the ventricle's contraction could not have arisen in the s.-a. node and must have arisen in the a.-v. node (see also Fig. 175).

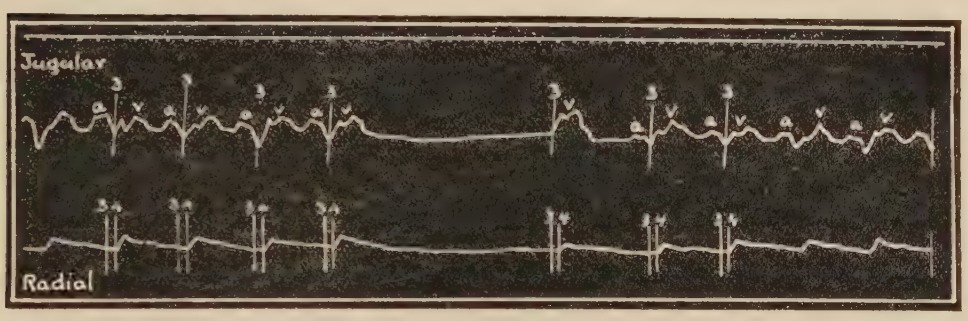


FIG. 37. Shows the temporary arrest of the whole heart from digitalis. The first beat after the long pause is a nodal beat, there being no auricular beat preceding it.

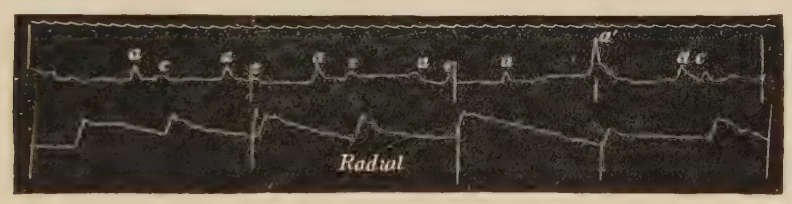


FIG. 38. Tracing from a case of partial heart-block, due to influenza. The large wave *a'* in the jugular tracing is due to the auricle contracting at its normal period at the same time that the ventricle contracts from impulses from the a.-v. node.

These examples are of rare occurrence, but they are of value in throwing light upon the peculiar function of the genetic system, and help us to get a better understanding of other forms of abnormal activity.

CHAPTER XXIV

Variations due to increased Excitability of the Genetic System

The extra-systole.
Clinical methods of investigating the extra-systole.
Experimental methods of investigating the extra-systole.
The genetic system and the extra-systole.
Different forms of extra-systole.
The electro-cardiogram of the extra-systole.
The irregularity produced by the extra-systole.

The extra-systole.—For a number of reasons I propose to deal with the subject of the extra-systole at some length, the main reason being that, although it is seemingly so simple a reaction and has been the subject of a great amount of research, both experimental and clinical, nevertheless its nature and its significance still elude recognition. Moreover, it is the most common of all the disorders of the heart-beat which the doctor meets in his practice, and it is frequently misunderstood. The consciousness of its presence may create alarm in the patients, which is often increased by the grave or guarded prognostications of the doctor.

The study of the extra-systole further helps to the understanding of the principle on which a sound prognosis can be based, and affords an illustration of the value as a guide of the application of the principle of the reflex arc in research.

The present-day knowledge of the extra-systole is practically based upon the result of experiment, and because there is a close resemblance between the experimental extra-systole and that found in nature, it is assumed that they arise in the same way. There is a fallacy here. Two men may be found at the bottom of a stair, and in this result there is a close resemblance, but there may have been a difference in the manner of getting there ; one may have walked down the stair, and the other may have been kicked down. There is a similar difference between the natural result and an experimental. While physiology has been content with the end result and with guessing at the manner of production, medicine requires that it should be known how the end result is brought about.

Clinical methods of investigating the extra-systole.—Over forty years ago I realized that I was profoundly ignorant of the nature of every symptom detected in my patients, and resolved to try to discover the mechanism of

the production of some of them. Irregularities of the pulse were of such frequent occurrence among my patients that I took up this subject. For a long time I was at a loss how to proceed. Many people had attempted to get this knowledge, both by clinical observation and by experiment, but at that time there was nowhere to be found any satisfactory method of investigation. In searching for a method an idea arose which has been extremely helpful. It came about in this way. While feeling the pulse, or studying the records of the pulse-beat on the sphygmogram, the question arose, 'What information did the pulse-beat yield ?' The answer was : it gave knowledge of the contraction of the left ventricle. The next question was, 'What were the other chambers of the heart doing when the ventricle contracted ?'

At that time it was known that the heart's contraction began somewhere in, or in the neighbourhood of, the superior vena cava, and from there the contraction spread like a peristaltic wave over auricle and ventricle. I thought, therefore, some light might be thrown on the problem by finding out what the auricle was doing at this particular pulse period.

This is the simple notion which has led far in the investigation of cardiac irregularities. To-day the guiding idea in the investigation of heart irregularities is 'What is the auricle doing ?'

For a long time I could not think of a method to get this knowledge. In the course of my inquiries into the mechanism of symptoms my attention had been arrested by the movements in the veins of the neck. Some years before this, when I was a resident in Edinburgh Infirmary, I was struck by some unusually large waves which were seen in a patient with a damaged heart. The only explanation given by my teacher when I asked him what they meant, was 'that they were a back-wash from the heart'. I managed to devise a method by which these movements could be recorded, by adding a Marey's tambour to a Dudgeon sphygmograph (Fig. 282). This has proved a singularly useful and convenient instrument, for it recorded the movements of the jugular and carotid in the neck at the same time as the radial pulse—the latter serving as a standard for the interpretation of the jugular. By this means it was often possible to get records of movements of the right and left ventricles and the right auricle, and sometimes even of the left auricle. Having acquired a knowledge of the interpretation of these records it occurred to me to use the method for recording heart irregularities, and in 1892 I obtained the first record in a human subject which showed the relation of the auricle to the ventricle, in what is now recognized as the ventricular extra-systole (Figs. 39 and 40).

In the interpretation of the tracing Fig. 40 I was able to demonstrate that the ventricle contracted earlier than, and independent of, the auricle, so that while the apex beat (upper tracing) showed an irregularity, the rhythm of the auricle was undisturbed (waves *a* and *a'*).

At that time I was working in an industrial town remote from any school of medicine or physiological laboratory, and had to find out my own explanations. This observation appeared to me to be so important that I

communicated with several physiologists, but they would say neither yea nor nay to my interpretations. At that time (1892) there was so little known of the heart-beat that no one had sufficient knowledge to appreciate such a record as Fig. 40. To the physician the condition was an intermittent pulse, and the curious sounds the heart gave out at the time of the intermission indicated that the heart had 'tumbled'. All that I could get from the physiologist was that the irregularity was a 'vagus pulse'.

Although doubt was cast upon the reliability of my method I knew that my tracings were reliable, for they recorded what I observed, and I published my results in 1893 and 1894, but they were ignored.

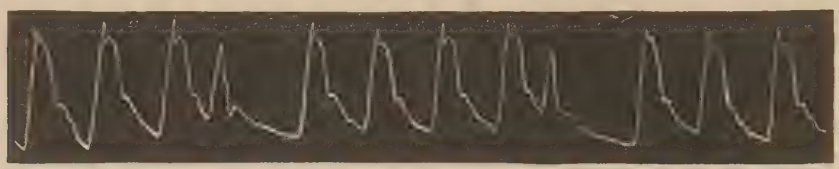


Fig. 39. The small beats are due to extra-systoles.

Experimental methods of investigating the extra-systole.—This began with Marey's experiments[1] upon the frog's heart. He showed by applying an electrical stimulus to the ventricle that during any period in the diastole it responded by a contraction which anticipated the normal contraction, and which he called the 'extra-systole'. Following this contraction there was a long pause. Engelmann (1894), working on the frog's heart, attributed

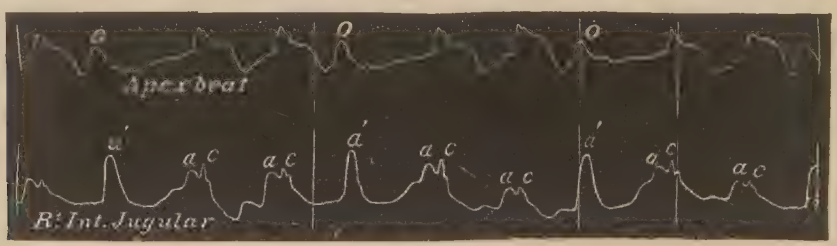


Fig. 40. Simultaneous tracings of the apex beat and of the jugular pulse, showing the rhythmical appearance of the auricular wave during the irregular periods in the apex tracing. The small beats *o o o* are extra-systoles of the ventricle. (This record was taken in 1892.)

the long pause to the 'ventricle' being in a 'refractory state' and not responding to the auricular impulse after the extra-systole. Cushny and Matthews (1897) demonstrated that by applying an electrical stimulus to the auricle or ventricle of the dog's heart, a premature or extra-systole of the auricle or ventricle followed. Cushny and Wenckebach (1899) recognized the resemblance of these results to the irregularities which occur in the human heart. From that date to the present the extra-systoles have been looked upon as arising from impulses in the auricle or in the ventricle, and the long

[1] Marey, *Circulation du Sang.*

pause that followed has been explained as due to a refractory state of the ventricle. What is meant by ' auricle ' and ' ventricle ' is the muscle element of these chambers, and the extra-systole is usually looked upon as being an evidence of a disturbance or disease of the myocardium.

Since the recognition of the genetic system, certain forms of extra-systole have been described as arising in this structure. But it is to be noted that the discovery of this system has had little influence in altering those conceptions formed at a time when it was not known to exist. When Tawara in 1906 published his book on *Das Reizleitungssystem*, a new conception was introduced whose full significance has not yet been appreciated. In reading Gaskell's early papers we can see that he was groping after the new idea, and realized that there was some factor at work which he dimly perceived. The discovery of this structure should have led to a thorough investigation of its function, but unfortunately this was not possible by experimental methods. Beyond some rather crude experiments when the bundle was cut or compressed, little could be done, and such experiments failed to reveal the real nature of its function, and the manner in which these functions are carried out. This is a problem which requires clinical methods and opportunities for its solution.

Here it is where both clinical medicine and physiology failed. It should have been recognized that this structure was so intimately associated with the heart's contraction that after its discovery every movement of the heart should have been reconsidered to find what part this system played. This was not done, and the result has been to incorporate the imperfect knowledge of this system with the conjectures and guesses by which the movements of the heart had been explained, with the consequence that the subject of the various movements of the heart has become more confused than ever.

The genetic system and the extra-systole. — After the genetic system was recognized I passed in review the great number of records of the jugular pulse I had taken, and I found a great many in which there was a delay between the contraction of the auricle and ventricle, shown by the lengthening of what I called the *a-c* interval. This sometimes went as far as to lead to the dropping out of ventricular systoles, and it was possible to trace this delay from the slightest degree through all stages up to complete heart-block. I also reviewed my observations of the extra-systole and recognized similar conditions present, and it occurred to me that the dropping out of the ventricular beat after an extra-systole was probably of the same nature as the dropping out of beats in partial heart-block, and in the first edition of this book I put forward the suggestion that the extra-systole was really due to a disturbance of the conducting system and not of the muscle. The view, however, was ignored, but the application of the principle of the reflex arc has led to a better understanding of the function of the genetic system, and demonstrates that extra-systoles can only arise from disturbance of this system.

Scrutinizing my records, a number of instances were found where it could be demonstrated that the dropping out of the ventricular systoles after the extra-systole was due to a refractory state of the a.-v. node or bundle.

The radial tracing in Fig. 41 shows a small beat, *r*, having the appearance characteristic of the extra-systole. It occurs earlier than usual and is followed by a pause longer than usual. The jugular pulse (upper tracing), taken at the same time as the radial, shows waves *a*, due to the auricular contraction. These features are represented in the intercalated diagram. Here the down-strokes in the space *As* represent auricular contractions, and in every instance excepting one this auricular contraction affects the a.-v. node (space *a-c*), and causes a contraction of the ventricle (space *Vs*). In the exceptional instance it is seen that the ventricular contraction represented by the small beat *r* occurs prematurely—before the auricular contraction and independent of it, and there is no ventricular beat following the next auricular contraction.

The description usually given of these events is that the ventricle had

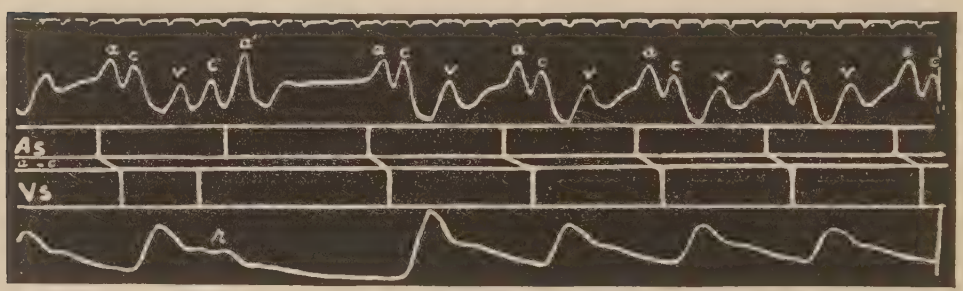


Fig. 41. Simultaneous tracings of the jugular and radial pulses, with an explanatory diagram intercalated, representing a ventricular extra-systole. There is a long pause after the extra-systole (*r*). In the jugular tracing and in the diagram this is seen to be due to the failure of the stimulus from the auricle (*a'*) to provoke a ventricular contraction.

contracted prematurely so that when the next stimulus for contraction from the auricle reached the *ventricle* it was refractory and stood still till another impulse came from the auricular systole, when the ventricle responded.

The expression that the *ventricle* is refractory does not describe the condition. If we study some of the instances where the extra-systole or premature beat is not followed by a pause but by a ventricular contraction,—the ventricle responding after every auricular systole,—we will find evidence for a better explanation of the events.

Fig. 42 is a tracing which shows in the radial pulse two extra-systoles or premature beats, *r*. The first of these is not followed by a long pause, while the usual long pause is seen after the second extra-systole.

In the jugular pulse the auricular waves *a* occur in regular sequence, but the carotid waves *c* and *c'* (corresponding to the radial beats) are not regular, the *c'* waves occurring before the *a* waves. Following the *a* wave, after the first extra-systole *c'* there is a *c* wave and a corresponding radial pulse-beat.

This wave and the corresponding radial beat are absent after the second extra-systole in the tracing.

The *a-c* intervals present (*A*) are of the same duration in every instance except in that which follows the first *c'* wave, when the interval is twice the normal length (A_1).

The interpretation of these events is that the bundle is concerned in the production of the extra-systole, so that when a stimulus comes down from the auricle the bundle is engaged in stimulating the ventricle, or the stimulus reaches the bundle before it has time to recover, and consequently it does not respond, and we get the characteristic pause after the extra-systole (as Fig. 41). When the bundle has sufficiently recovered (as in Fig. 42) so that the impulse gets through the bundle, it finds the ventricle capable of giving a forcible and effective contraction, as shown by the large beat which follows the extra-systole *r*.

From this line of reasoning we see that it is not the ventricle that is refractory but the bundle.

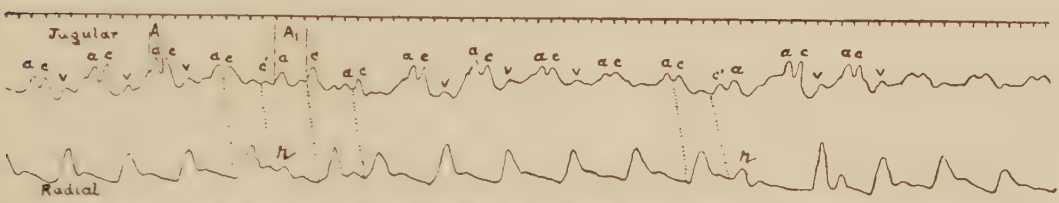


FIG. 42. The radial tracing shows two extra-systoles (*r*); after the second *r* there is a long pause. After the first there is not a long pause, showing that the extra-systole here is interpolated. In the jugular tracing the *a-c* interval (*A*) is normal, but the *a-c* interval that follows the first extra-systole *c'* is greatly increased (space A_1).

These events are brought out also in the electro-cardiogram. Fig. 43 is a record of the electro-cardiogram, the movements in the neck and the radial pulse *R'*, *c'*, and *r* are due to the same premature or extra-systole. They are not followed by a long pause but by a normal ventricular contraction. The *P-R* and *a-c* intervals following the extra-systoles (*R'* and *c'*) are longer than the other *P-R* and *a-c* intervals.

Figs. 44-46 show a number of these interpolated extra-systoles, and all bear out these features. Fig. 47 is a tracing taken at the same examination as Fig. 46 was obtained.

Different forms of extra-systole.—The extra-systoles show differences according to the part of the genetic system from which they arise.

The 'auricular' extra-systole is recognized by the auricular contractions occurring prematurely, as in Figs. 48-51, the nodal extra-systole by the simultaneous and premature contraction of auricle and ventricle, as in Fig. 53, and the 'ventricular' extra-systole which arises in the branches within the ventricle by the fact that it precedes or occurs at the same time as the normal auricular contraction, as in Figs. 41, 42, 47, etc.

The electro-cardiogram of the extra-systole.—The foregoing explanation of

the origin of the disturbance which results in an extra-systole is supported by the electro-cardiogram. Fig. 52 is an example of a fairly common form of

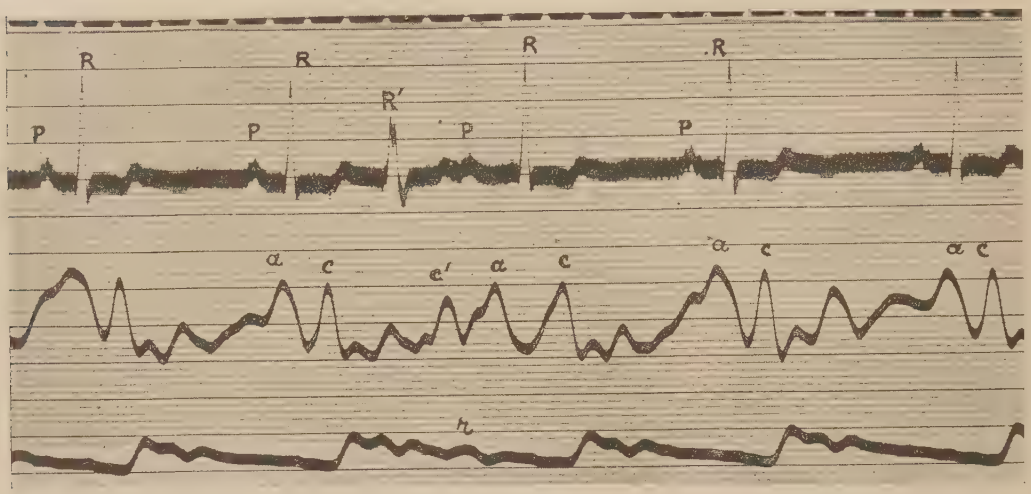


Fig. 43. Shows an electro-cardiogram and tracings of the jugular and radial pulses. Each of them shows one extra-systole (R', c', r). The P-R interval and the a-c interval are increased after the extra-systole (J. Parkinson).

the extra-systole where the disturbance arises in some branch of the genetic system within the ventricle, producing a very small pulse-beat or none at all. The electro-cardiogram in Fig. 43 shows an extra-systole probably arising in

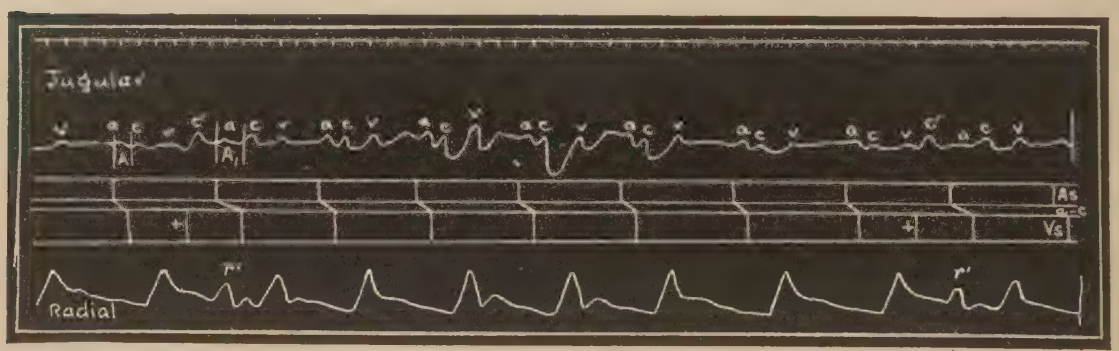


Fig. 44. Simultaneous tracing of the jugular and radial pulses, showing the interpolation of ventricular extra-systoles (c', c' and r', r') represented in the diagram by the down strokes + +. The down strokes in the space As represent the auricular waves (a) in the jugular, and the down strokes in the space Vs represent the carotid waves c, and the slanting lines connecting them represent the a-c interval. Note increased a-c interval A_1.

the bundle, from its shape and the large pulse-beat. Fig. 54 shows the extra-systoles arising in the auricle when the auricular wave is inverted. In Figs. 43 and 54 it will be seen that the ventricular complex is in striking contrast to Fig. 52.

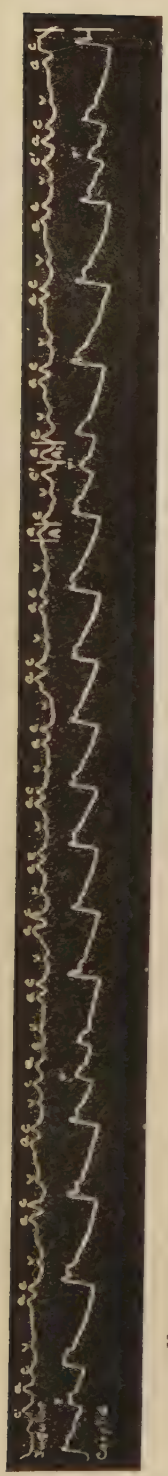


Fig. 45. Shows the occurrence of interpolated systoles c', r' with a prolonged a-c interval (A_1) after the extra-systoles c'.

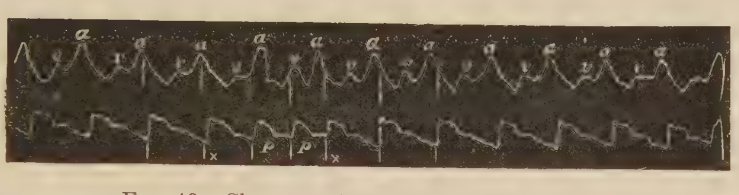


Fig. 46. Shows one interpolated extra-systole.

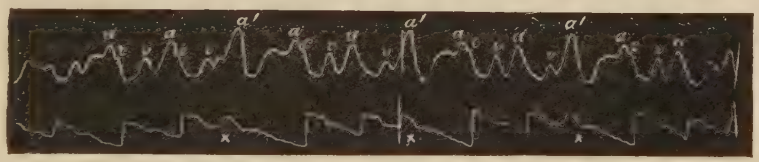


Fig. 47. Taken the same time as Fig. 46, showing extra-systoles followed by a long pause.

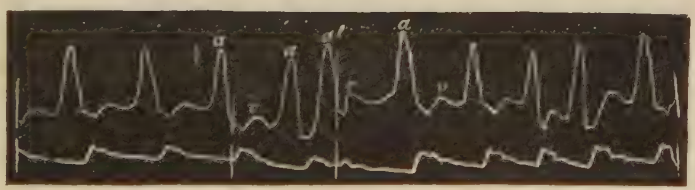


Fig. 48. Simultaneous tracings of the jugular and radial pulses, showing a large wave (a') due to an extra-systole of the auricle.

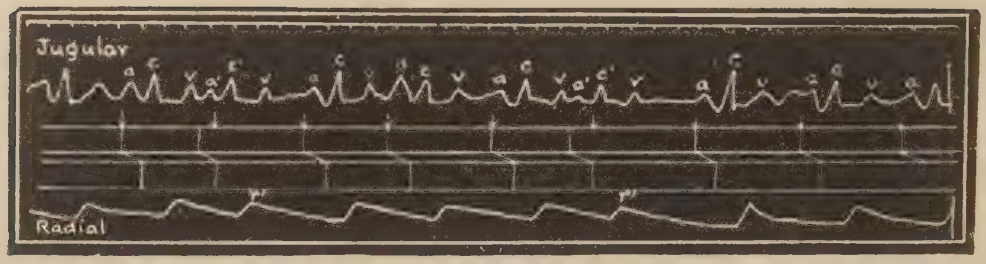


Fig. 49. Shows auricular extra-systoles (a') followed by ventricular contractions (c', r'). The arrows in the diagram represent the sinus stimulation, and the long pauses after the extra-systoles are seen to be due to the fact that the auricle did not respond to the sinus stimulation.

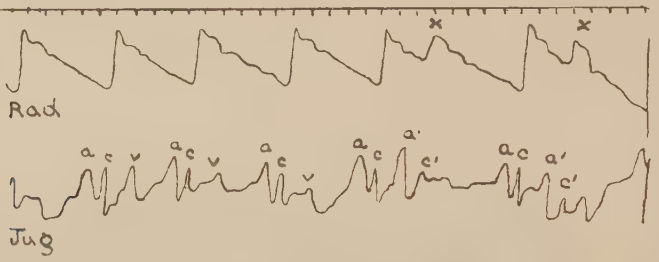


Fig. 50. Shows two premature or extra-systoles of auricular origin (×). The waves c' in the jugular tracing occur at the same time as the small premature beats (×) in the radial tracing, and are therefore due to the carotid. These are preceded by premature waves a' due to the auricle.

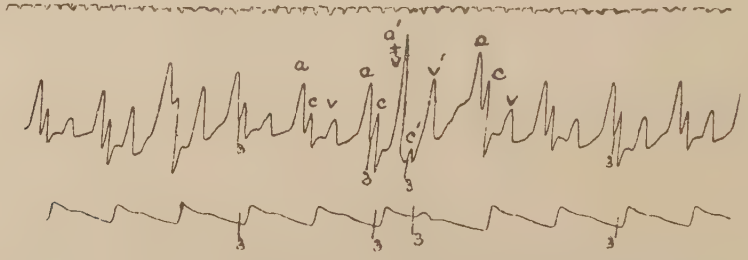


Fig. 51. Shows an auricular extra-systole—the premature wave a' falling on the wave v of the previous ventricular systole.

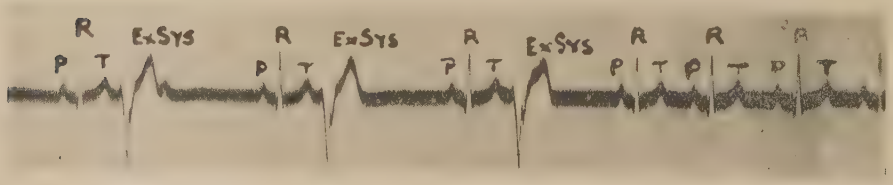


Fig. 52. An electro-cardiogram showing three extra-systoles (*ExSys*) with normal beats (*P R T*).

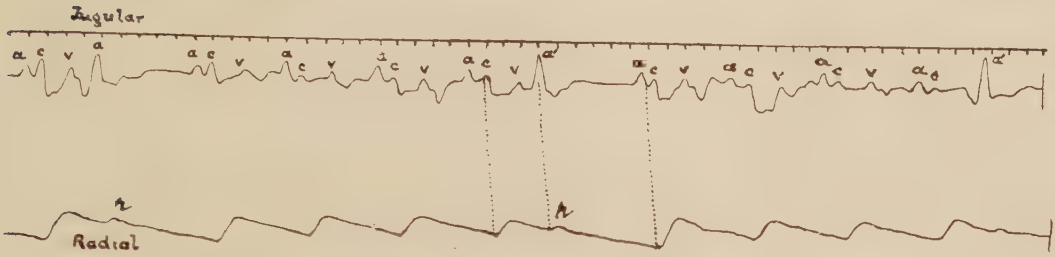


FIG. 53. Shows a nodal extra-systole—the auricle (a') and the ventricle (r) contracting prematurely and together.

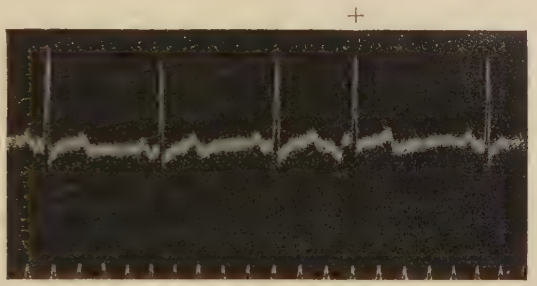


FIG. 54. Shows an auricular extra-systole (+) with an inverted auricular wave Compare with Fig. 34. (Rothberger.)

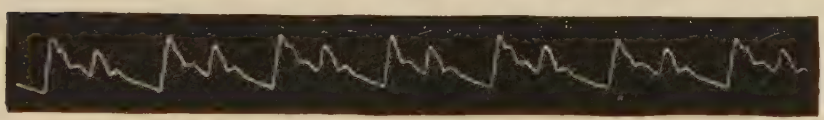


FIG. 55. Pulsus bigeminus due to an extra-systole occurring after each normal beat.

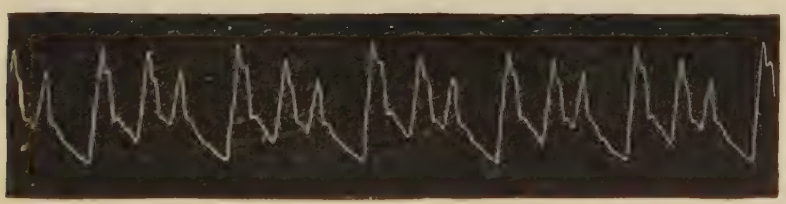


FIG. 56. Extra-systole occurring after every two normal beats.

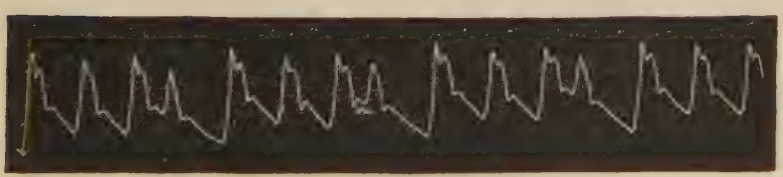


FIG. 57. Extra-systoles occurring after every three normal beats

The irregularity produced by the extra-systole.—In the majority of instances the extra-systole breaks in upon the normal rhythm at irregular intervals, sometimes rarely and sometimes frequently. When at rare intervals (Fig. 39) it is easy to recognize by its solitary appearance, and when at more frequent

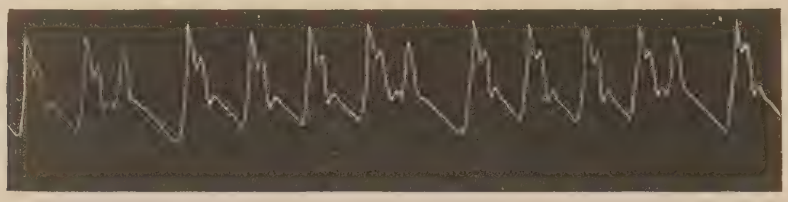


Fig. 58. Extra-systoles occurring after every four normal beats.

intervals it may assume the characters of a disorderly rhythm. Occasionally it may appear with remarkable constancy. In some instances each normal beat is followed by an extra-systole, as in Fig. 55. Such irregularity of this sort may be present in individuals for weeks and months. An extra-systole may occur regularly after every second, third, or fourth normal beat, as in Figs. 56, 57, and 58.

The reason for this periodicity of the extra-systole cannot be satisfactorily explained in our present state of knowledge.

CHAPTER XXV

Variations due to increased Excitability of the Genetic System (*continued*)

The size of the extra-systolic pulse-wave.
Comment upon the origin of the extra-systole.
Clinical diagnosis.
Prognosis.
Treatment.

The size of the extra-systolic pulse-wave.—Variations in the size of the radial pulse have not received that consideration which they merit. They afford a useful index of the efficiency of the individual contraction of the left ventricle. They vary in size according to the site of origin of the impulses. While normally the impulses are started in the sino-auricular node, they may start at different parts of the conducting system. The efficiency of the contractions of the ventricle varies with the part of the genetic system in which the impulses arise.

The normal contraction is graded and regulated in a manner that favours the expulsion of its contents. This is brought about by the manner in which the genetic system stimulates the fibres of the muscle to contract in a particular sequence. When the stimulus for contraction arises in one of the branches of the genetic system within the ventricle, the contraction of the fibres in the normal sequence does not occur, and in consequence the contraction may be less effective in emptying the contents of the chamber. It is readily conceivable that the extra-systole starting at different parts will spread the contraction in different directions so that the contractions may not all equally facilitate the expulsion of the ventricular contents; in consequence there occurs that variation in the size and in the absence of a pulse-beat which is a common feature of the extra-systole.

When the size of the pulse-beat approaches the normal it may be taken as an indication that the disturbance has taken place in some part of the genetic system before the bundle has broken into its branches. In these instances the impulses enter the ventricle by the bundle, so that the contraction of the muscle fibres follows the normal sequence. It is for this reason that the auricular and nodal extra-systoles, that is where the disturbance has arisen in the genetic system in the auricle, or in the a.-v. node, are usually fairly large pulse-beats (Figs. 47-51 and 53).

Hitherto the variations in the size of the beats have been attributed

mainly to the occurrence of ventricular contraction at an earlier period when the contents of the ventricle were less, or when the ventricle was so exhausted that the contraction was weak. This explanation does not cover the facts. If such were the case it would be expected that the longer the pause before the extra-systole the larger would be the pulse-beat, while the shorter the pause, the beat would be smaller. But we find, as seen in Figs. 59 and 60,

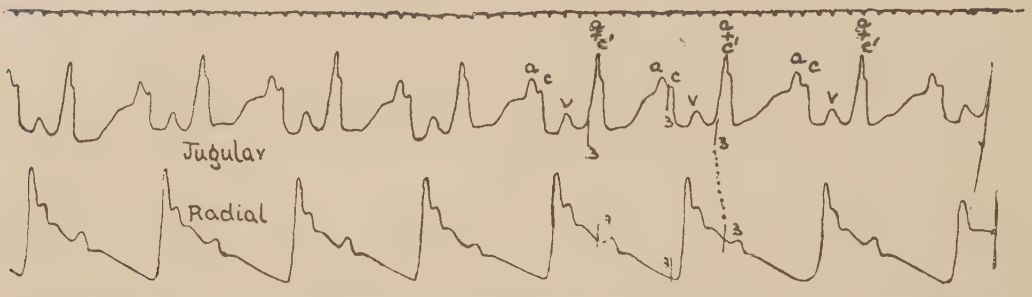


FIG. 59. Shows a small extra-systole on the radial tracing after a fairly long pause. The jugular tracing shows a large wave $a+c'$ occurring at the same time as the extra-systole; showing that auricles and ventricles contracted together.

that small beats may occur when the preceding pause is little shorter than the normal interval, and even the contraction of the ventricle may be so ineffective that its force does not reach the radial pulse (Figs. 61, 62, and 63). In Figs. 59-62 the extra-systole falls at the same time as the auricular

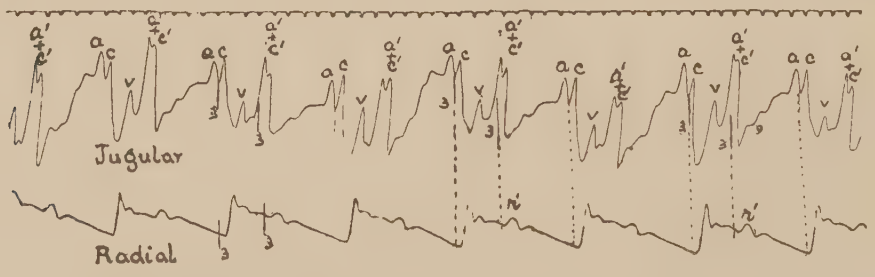


FIG. 60. The radial tracing shows a premature beat or extra-systole after each normal beat. The jugular tracing shows a large wave $a'+c'$ occurring at the same time as the extra-systole; showing that the auricles and ventricles contracted at the same time.

systole. On the other hand, fairly large pulse-beats may follow very short pauses, especially in auricular or nodal extra-systoles (Figs. 49, 50, 53). Such facts support the view just expressed that the size of the beat depends upon the sequence of contraction of the muscle fibres.

Another evidence is seen in the instance of an interpolated extra-systole. The extra-systole may be of small size compared with the normal contraction that follows, and the pause before the extra-systole may be longer than that before the normal contraction (Fig. 44).

Comment upon the origin of the extra-systole.—It is worth while to review the facts just recorded to appreciate the reasons for attributing the extra-systoles to a disturbance of the genetic system.

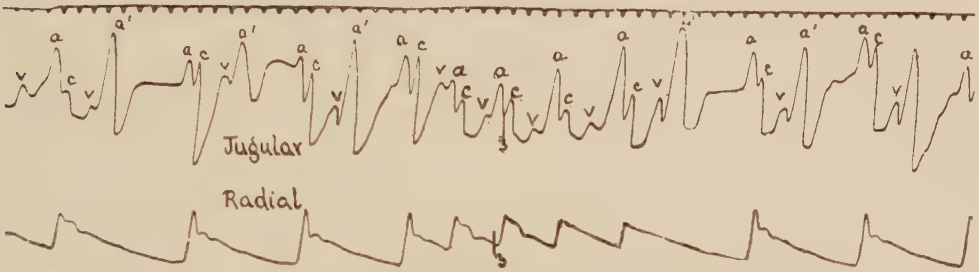


FIG. 61. Shows periods of normal heart action varying with periods in which the extra-systoles occur. There are two periods of irregular pulse-beats (three pulse-beats in each period). The jugular pulse shows that, after each of the pulse-beats, there is a large wave *a'*, due to a premature contraction of the auricle. On account of the size of the wave, it can be inferred that the ventricle was also in contraction, so that the auricular contents could not go forward into the ventricle, but were sent back into the veins.

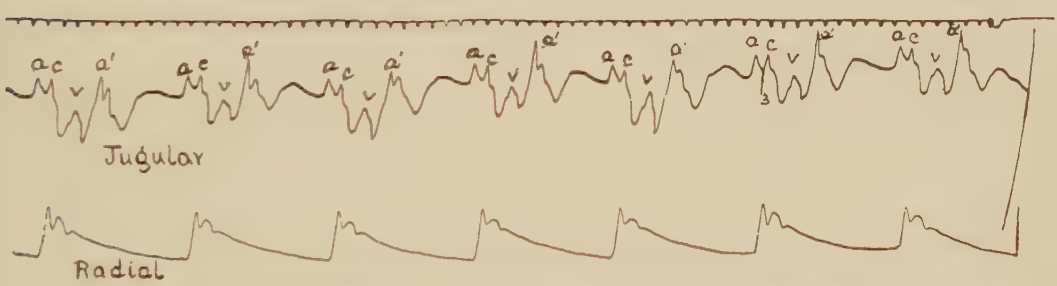


FIG. 62. The jugular pulse shows large beats *a'* during the long pause in the radial, indicating that the auricles and ventricles contracted, but that the ventricular contractions were too weak to produce a wave in the radial pulse sufficiently large to affect the sphygmograph.

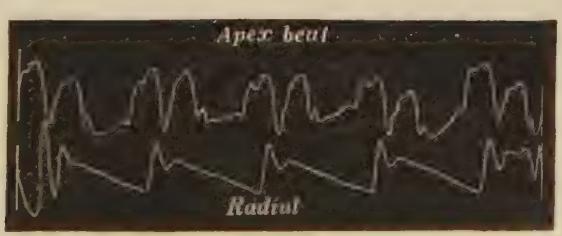


FIG. 63. Simultaneous tracings of the apex beat and of the radial pulse. There are two beats of the apex to one radial pulse.

In the first place, it is in agreement with the great principle that the co-ordinated contraction of muscle cells is always due to impulses from controlling structures. On p. 56 there were described three forms of muscle contraction.

When we consider the various forms of extra-systole, we can recognize that as there is an effective contraction of the muscle the impulses must be conveyed to the muscle by a special structure. The extra-systole is in this sense an effective contraction, even although in some instances the output from the ventricle is small. The reason for that has been explained. In the second place, in the study of the pause after the extra-systole and the interpolated extra-systole there are distinct evidences that the long pause is due to the failure of the genetic system to convey the impulse for contraction. In the third place, the evidences from graphic and electro-cardiographic records show that the various forms of extra-systole are due to disturbances in various parts of the conducting system.

The view that has been held up to the present that the extra-systole arises in the ventricle or in the auricle, does not specify the structure in which the original disturbance takes place. This can only be the genetic system or the muscle fibres, and so far there has been adduced no evidence demonstrating that muscle fibres can originate a stimulus that would produce an extra-systole. When muscle fibres contract independently of the genetic system the contraction takes the form of fibrillation.

The statement that the long pause is due to a refractory state of the ventricle is too vague and indefinite. What is generally meant by the term ventricle is the muscle, and again we have no evidence that a muscle can become refractory. It can be reasoned that, like all other cells, the muscle cell is bound to have a refractory stage, but so far the evidence does not show that it could be so long as to result in an inability to respond to the auricular contraction which follows the extra-systole. The refractory state is manifestly due to the genetic system.

The significance of the origin of the extra-systole will be found in dealing with the prognosis of extra-systole.

Clinical diagnosis.—In the vast majority of individuals the extra-systole is easy to recognize. When occurring at rare intervals it appears as an intermission in an otherwise regular pulse. Usually it is not perceptible to the finger, but the apex beat may show its presence. At one time the presence of an apex beat during this pause (as in Figs. 40 and 63) was taken as convincing evidence that the right heart contracted while the left stood still. Careful records, however, showed that both ventricles participate in producing the extra-systole. Further evidence of the presence of an extra-systole can be obtained by auscultation, when the first and second sounds produced by the extra-systole are heard to occur as two slight, short, sharp sounds (Figs. 64 and 65). The sounds are often close together, sometimes one is wanting, and we hear but one muffled sound.

Many patients are conscious of its presence either by a peculiar sensation in the chest, or it may produce some other sign, as a slight cough. The patient may be conscious of the pause in the heart, and this may cause a certain amount of mental distress, some patients thinking that the heart is about to stop. After the pause some patients are conscious of the big beat, and the shock which it produces also causes mental anxiety.

From the patient's own description we can often recognize that it was an extra-systole which gave rise to these sensations.

Prognosis.—As one of the objects of this book is to call attention to the importance of prognosis and the principles which should guide a search for

Fig. 64. Diagram representing the sounds of the heart with occasional extra-systoles.

prognosis, the extra-systole forms one of these examples which lend themselves to a demonstration of the principles.

For a long time the intermittent pulse was looked upon with grave suspicion, and even to-day many authorities regard it seriously. Some examiners for

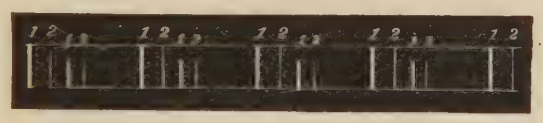


Fig. 64a. Diagrammatic representation of the sounds of the heart in a case of such rhythmical irregularity as is represented in Figs. 59-63.

life insurance still reject people or penalize them on account of an extra-systole. Other examiners consider it a bar to entering the services, while great numbers of patients are compelled to submit to different forms of treatment and restrictions because of the presence of an extra-systole.

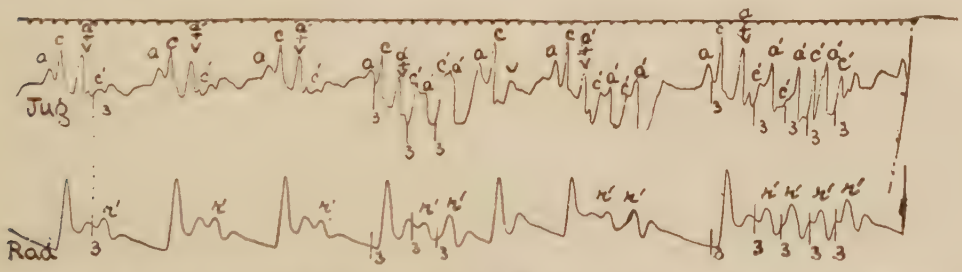


Fig. 65. Multiple auricular extra-systoles (r'). One auricular extra-systole occurs after each normal beat in the first part of the tracing, and several in the latter part of the tracing.

When in the first edition of this book I called attention to the fact that the extra-systole was without any prognostic significance, my statement was not accepted, many physicians adopting the view that the extra-systole required a 'guarded prognosis'; and even yet some of our later cardiologists think that this view of mine is too general, and that it may have a grave significance. This hesitation and doubt is simply due to the fact that it is not yet recognized

what steps to take to assess the value of a symptom, nor is the difference between a symptom due to a cause in itself of little moment and a symptom which may be associated with conditions of grave moment sufficiently appreciated.

For over forty years I have kept records of patients with extra-systoles. They were found to be rare in the young, and a little more frequent about middle age. Over 50 years of age many people exhibit them, while probably every person over 60 exhibits them at one time or another, or continually. We recognize in this review an occurrence similar to what happens to people with grey hairs, and so far as I have seen, I would attach no more gravity to the one sign than to the other.

If cardiac impairment is found associated with extra-systoles, then the prognosis depends upon the condition of the heart which leads to the impairment, and not upon the extra-systole, which, at the worst, indicates but a coincident change.

When I reflect upon the enormous number of people in whom I have found extra-systoles, a great many of whom I watched pursuing laborious occupations with no detriment to their health, and encountering illness which imperilled their lives, I cannot see what better evidence we could find for forming the opinion that I give here. On the other hand, it is surely incumbent on those who dispute these conclusions to produce evidences of the danger which arises from the extra-systole with the history of illustrative cases. So far, no evidence has been produced.

I have, of course, seen many people become ill who had extra-systoles, and even developed them during their illness, and these illnesses have terminated fatally, but there was no reason for assuming that the extra-systole was in any sense a sign of danger ; all that could be said was that the conditions which produced danger produced the extra-systole, as it produced an increased pulse-rate, perspiration, or other sign, innocent in itself.

Up till recently the only method by which this kind of knowledge could be obtained was by watching individual patients for years to see what happened. With the recognition of the principles which govern heart failure and the heart's action, a better method has lately emerged—described on pp. 23-24. We can distinguish phenomena produced by the different structures of the heart, and each of these structures has a different value in the production of heart failure ; some have little relation to the actual work of the heart. The genetic system merely regulates the contraction of the heart, and disturbances of it can have little effect upon the function of the heart except when they cause a great increase or a great decrease in rate. As the occasional extra-systole does neither of these things, *a priori* it could have been reasoned that it could have no crippling effect upon the heart's functions.

So long as the extra-systole was supposed to be due to the ' ventricle ', and by the ventricle was meant the heart muscle, it was looked upon as evidence of a damaged myocardium, and therefore considered to be a possible sign of some gravity ; but as it is produced by a disturbance of the regulating system, in itself it can have no grave significance. This line of reasoning brings

the extra-systole into line with the other disturbances of the conducting system.

This being so, the question arises, seeing that it is not an evidence of muscle impairment, does its presence embarrass the muscle in its work? This is a question which can easily be answered. As the integrity of the circulation depends upon the efficiency of the heart muscle, can the extra-systole impair the efficiency? Seeing that the impaired function of a muscle is due to exhaustion, the long pause after the extra-systole favours the restoration of the heart muscle, so that what energy may have been spent by the ineffective extra-systole is partly made good by the long rest which the ventricle gets.

Further, the extra-systoles tend to occur when the body is at rest and the heart is working well within its capacity, at a time when the heart has plenty of surplus energy in reserve. As a rule, when bodily effort is made, the extra-systoles disappear (see Observation 2, Fig. 83*a*).

This view agrees with experience, for I have never seen an individual whose impaired response could be attributed to extra-systoles.

The only possible danger might be that the nature of the disturbance which results in an extra-systole might be of a progressive kind, and lead ultimately to a grave condition. The long periods during which I have watched patients show such a fear to be groundless, though the frequency of occurrence of the extra-systole increases with advancing years.

It may therefore be stated that when the extra-systole is the only abnormal sign, the prognosis is a favourable one; and where it is associated with other signs, the prognosis is to be based upon these other signs.

Treatment.—The patient is usually unconscious of the presence of these extra-systoles, but may be alarmed by his doctor telling him that he has an intermittent or irregular heart. The patient may be conscious of the pause, and may feel frightened lest the heart should stand still, or be painfully aware of the big after-beat. As these extra-systoles frequently occur in the night, when the heart is otherwise acting slowly, patients, particularly neurotic individuals, are greatly distressed. As the presence of extra-systoles and the patient's consciousness of them cause great mental distress, it is always necessary in treatment to take the whole mental state into consideration. The alarm is often aggravated by the inability of the doctor to recognize the significance of the irregularity, and by his subjecting the individual to a long course of treatment, or sending him to some health resort which advertises itself as a cure for heart affections. As all attempts at cure are often unavailing, many individuals go through life obsessed with the idea that they have a serious affection of the heart. The first duty of the physician is clearly to recognize the nature of the trouble, and to relieve the patient's mind so far as this irregularity is concerned. There is absolutely no cause for alarm. I frequently see members of our profession greatly distressed because they themselves have detected this irregularity. They have sought in text-books for an explanation of their trouble, and, finding nothing but vague statements,

they have become oppressed by the mystery of their symptoms. The intelligent explanation of the nature of the irregularity has at once dispelled this fear. So it is necessary that we should, with confidence based on knowledge, reassure the patient in the first instance as to the innocent nature of the complaint. When some digestive disturbance, or such articles as tobacco, tea, or coffee, predispose to extra-systoles, the alteration of the diet and the omission of the offending article are quite sufficient to stop their occurrence.

In neurotic people, when their occurrence causes distress, especially during the night, ammonium bromide (20 grains two or three times a day) usually gives relief after a few days. In the vast majority of cases drugs are of no use. Many drugs, such as digitalis, have a reputation for stopping them, and it is quite possible that it and other drugs may stop them as easily as others start them ; but it will be found that there are periods during which the extra-systoles are of frequent occurrence, and long intervals during which they are absent, and the cessation of the extra-systoles is, in these cases, quite independent of the drug. When they persist for months and years, no form of treatment appears to be of use in stopping them. I have carefully pushed many drugs in these persistent cases ; and I have seen individuals who have been subjected to all sorts of treatment ; but I have been unable to find any drug or method which was effectual in preventing their occurrence. Many people are entirely free from them while they take plenty of healthy exercise in the open air, but have them when leading a somewhat sedentary life, so exercise in the fresh air should be recommended to such patients.

CHAPTER XXVI

Variations due to increased Excitability of the Genetic System (*continued*)

Paroxysmal Tachycardia

The nature of the attacks.
The different forms of paroxysmal tachycardia.
The small beats.
Clinical diagnosis.
Prognosis.
Treatment.
Observation 2.

The nature of the attacks.—In the description just given of the extrasystole it was shown that its production was due to some part of the genetic system being rendered unduly excitable. The result was the breaking in on the normal rhythm by a premature contraction of the ventricle, or of the auricle and ventricle. This disturbance of the normal rhythm is limited to one beat.

There are conditions in which there may be several beats or a series of beats lasting for a considerable length of time. The characteristic features of such attacks are the suddenness of onset and the suddenness of offset, and the increased rate of the pulse. The reason for suddenness of onset and offset is simply because the excitable part starts throwing out impulses before the s.-a. node, and as soon as it ceases to do this the s.-a. node resumes control.

During the interval between onset and offset the rhythm is always regular while the rate is usually greatly increased.

These are the chief features of the group of abnormal rhythms to which it is proposed to restrict the term paroxysmal tachycardia. They differ in their origin and in their nature from paroxysmal attacks with auricular fibrillation and flutter for reasons that will be understood when dealing with these two conditions. They also differ from attacks of increased rate of the heart due to increased excitability of the s.-a. node. This latter form of increased heart-rate may begin somewhat suddenly, but not with that abruptness of onset which characterizes paroxysmal tachycardia, for here the disturbance is not due to a part of the conducting system which anticipates the stimulus from the s.-a. node, but arises in the s.-a. node itself. Again, its subsidence is gradual, the excitability of the s.-a. node gradually diminishing.

The different forms of paroxysmal tachycardia.—The different forms depend, like the extra-systole, upon the part which is disturbed. If it affects the genetic system in the auricle there results a tachycardia in which the auricle participates. Such attacks vary in duration, some ranging from the single extra-systole to two, three, or four (Fig. 65) up to short and long runs, as in Figs. 66 and 67. In Fig. 66 there is shown a characteristic short attack,

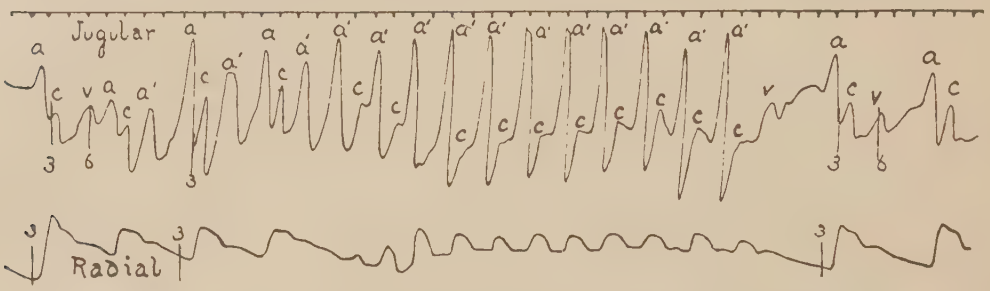


Fig. 66. Shows a short attack of paroxysmal tachycardia. The waves *a'* are due to contractions of the auricle arising from impulses discharged from the genetic system in the auricle other than the s.-a. node. The attack begins abruptly and ends abruptly.

showing the abrupt onset and the abrupt offset. In Fig. 67 there is a termination of a long attack which had been going on for many minutes.

These tracings show large auricular waves in the jugular, and a radial pulse-beat follows each auricular contraction. The height of the wave is due to the contraction of the auricle occurring before the preceding ventricular

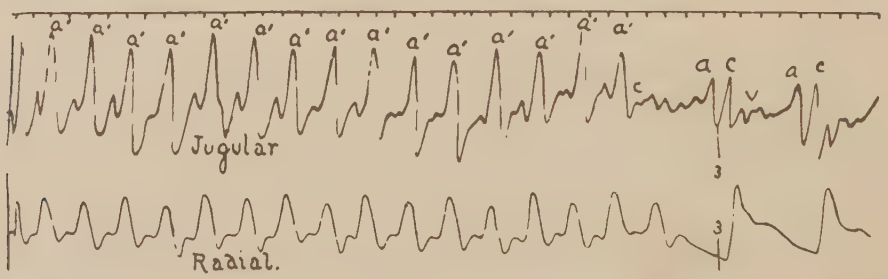


Fig. 67. Is from the same patient as gave Fig. 66, and shows the termination of a long attack.

systole has ceased, and not due, as in the next series of cases, to the auricle and ventricle starting to contract at the same time.

Fig. 68 shows electro-cardiographic records from a patient with this form of tachycardia. It will be seen that here also the auricular contraction (*P*) is followed by a ventricular systole.

It may be as well to point out that the resemblance between the graphic records in Fig. 66 and 67 and the electro-cardiogram, Fig. 68, is superficial, the large waves *a'* in the jugular being due to the auricle—the great size being due to the reason already given—while the large wave in Fig. 68 is due to the ventricle.

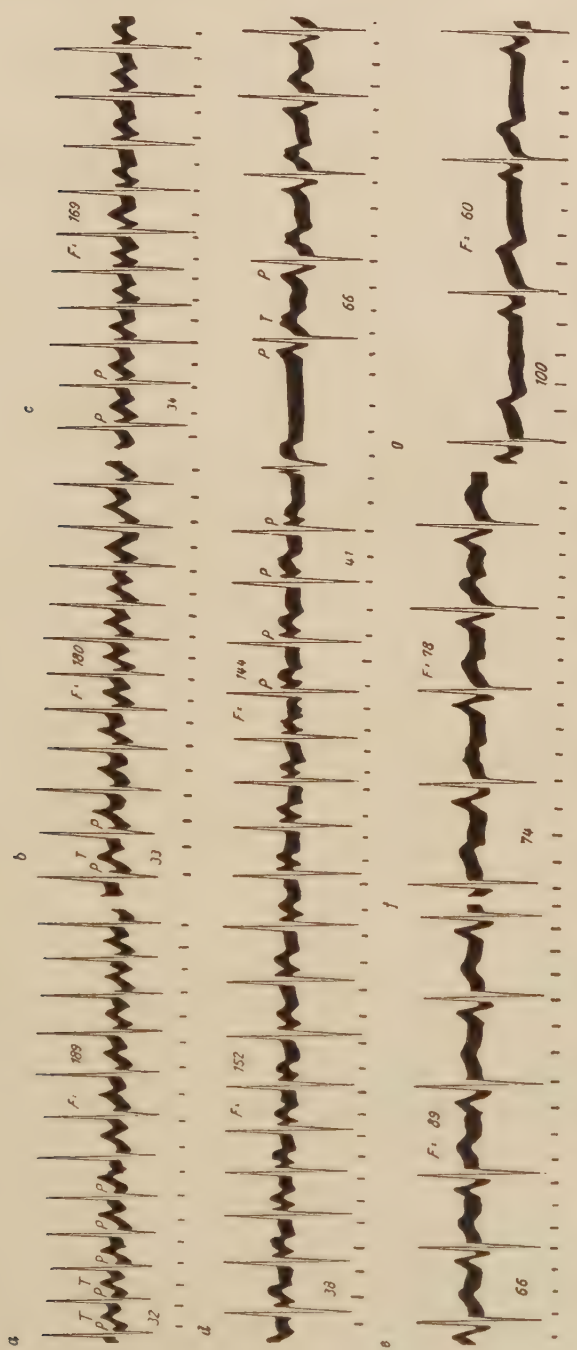


FIG. 68. Is an electro-cardiogram of an attack of auricular tachycardia showing the effects of quinidine. In the first part the rate was 189 per min., taken before administration of quinidine. The second part (*b*) was taken immediately after an intravenous injection of quinidine, the rate 180. Period *c* half a minute later, the rate 169 per min. Period *d*, one minute later, the rate has fallen to 152-144, and the normal rhythm becomes established. The rate gradually fell during the normal rhythm from 92 to 78. Twenty-four hours later, period *g*, the rate had fallen to 60 per min. (Singer and Winterberg quoted from Rothberger's German translation of the third edition of this book, Fig. 189, p. 296.)

Tachycardias may arise from disturbances of the a.-v. node and are shown in Figs. 69-74, all from the same patient. Here again the attacks range from a single extra-systole to two or three together up to short and long runs. The first of these tracings, Fig. 69, shows the jugular and radial pulses when the

Fig. 69. Shows the normal rhythm.

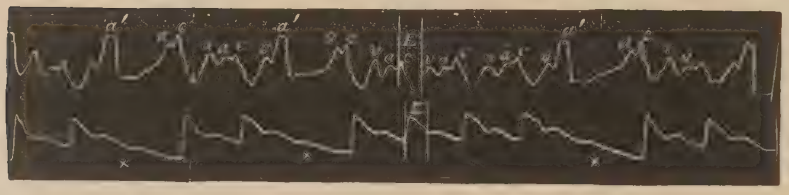


Fig. 70. The simultaneous tracings of the jugular and radial pulses during irregular action of the heart. The auricular waves *a'* are premature, and occur at the same time as a ventricular extra-systole, which is shown by the increased size of the auricular waves *a'*. Here the extra-systole is nodal in origin.

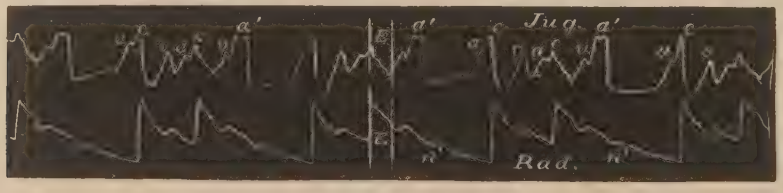


Fig. 71. Shows the same as Fig. 70.

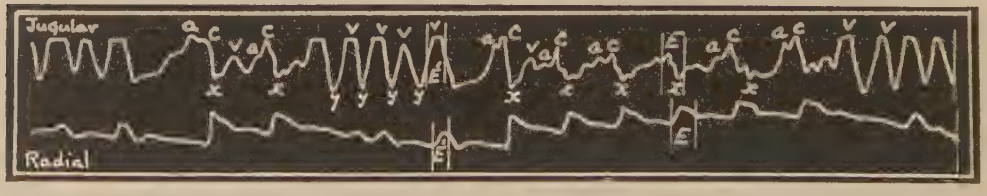


Fig. 72. Taken at the same time as Figs. 70 and 71, and shows groups of nodal extra-systoles.

heart is beating in a normal manner. The next two tracings show frequent extra-systoles where both the auricle and the ventricle contracted prematurely and at the same time, as shown by the wave *a*—being larger than the other waves. The next tracing shows the regular rhythm interrupted by a short series of these premature beats. The last two tracings show longer attacks interrupted by normal beats.

The prematurity of both auricle and ventricle indicates that the impulses for contraction arose probably in the a.-v. node, and the size of the *a* wave is due to the usual cause when auricle and ventricle contract together—see Figs. 70 and 71.

Tachycardias may arise from disturbance of the branches of the a.-v. bundle within the ventricle. The pulse-beats then have the same character as that of the 'ventricular extra-systole'. The electro-cardiograms are also identical, as will be seen by comparing Fig. 75 with Fig. 52.

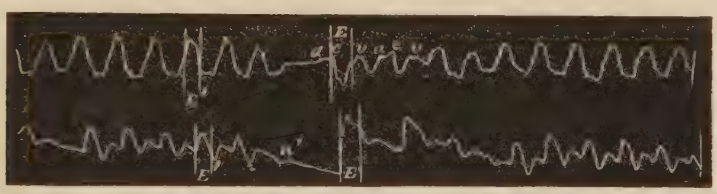


Fig. 73. Shows two normal beats interrupting a long series of nodal beats.

Fig. 74. Shows the same as 73.

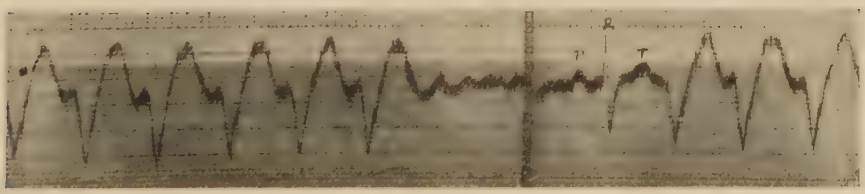


Fig. 75. An electro-cardiogram showing a normal beat *P R T* interrupting a series of abnormal ventricular contractions. These are of the same kind as the ventricular extra-systole.

This form of paroxysmal tachycardia also varies from the single extra-systole to longer periods. It is seldom prolonged beyond 10 or 20 beats at one period. There may be a long series of such periods interrupted by normal beats, as in Figs. 76, 77, and 78. In Fig. 78 the pulsus alternans is present. A few minutes after these attacks passed off the normal beats also showed the pulsus alternans (Fig. 79).

These cases are relatively rare. In the majority of instances the attacks are transient, although they may recur at frequent intervals. One patient who had this condition for over a year evidently fairly constant, suffered a good deal of distress chiefly due to breathlessness and pain in the

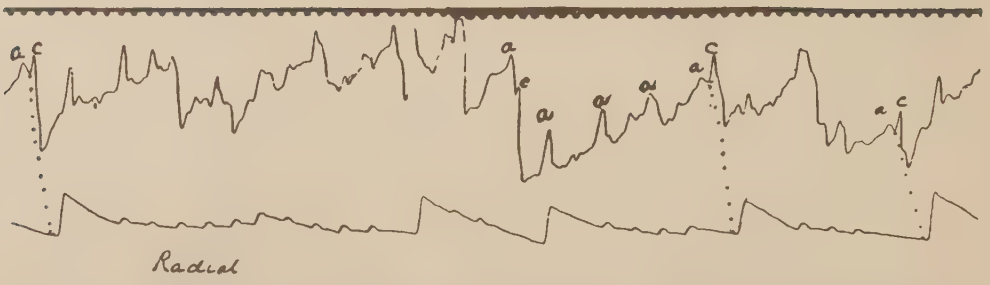


FIG. 76. Simultaneous tracings in the neck and radial pulse taken at the same visit as Fig. 75, showing the small, rapid beats in the radial corresponding to those in the electro-cardiogram. The waves *a* from the neck are due to systoles of the auricle, which have no relation to the small and more numerous radial beats.

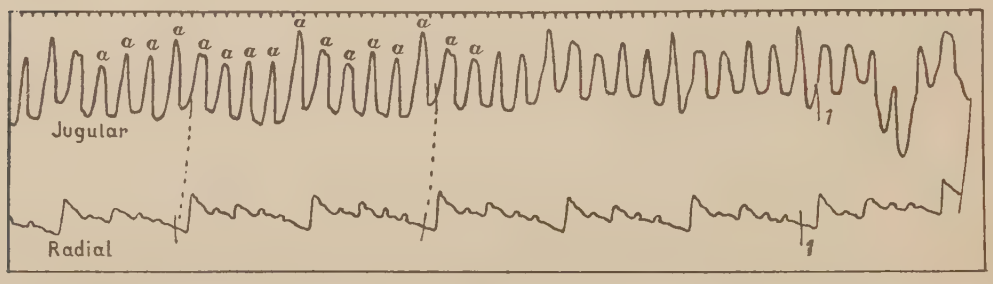


FIG. 77. Simultaneous tracings of the jugular and radial pulses during an attack of paroxysmal tachycardia. The waves *a* in the jugular tracing are due to the contraction of the right auricle, and are quite regular, in contrast to the irregular rhythm shown in the radial tracing. The radial beats occur in groups of five, the first of which is a normal beat and arises in response to an auricular contraction, while the other four beats arise in the genetic system of the ventricle independent of the auricular beats, and at a slightly more rapid rate.

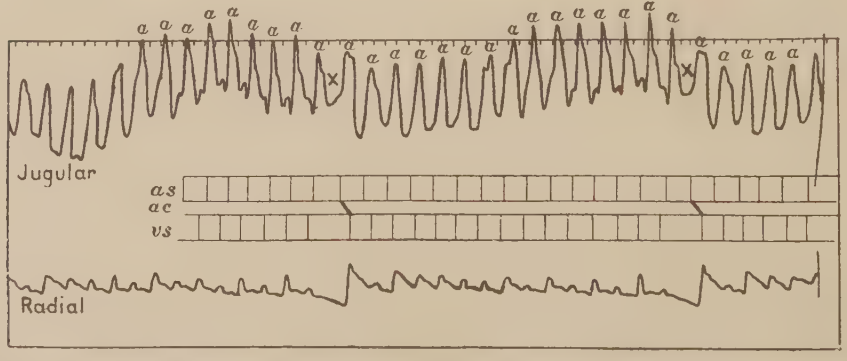


FIG. 78. Is part of the same tracing as Fig. 77, and shows the same events, excepting that the run of abnormal beats is longer. The radial tracing shows the pulsus alternans occurring in the abnormal beats. The interpolated tracing shows the relation of the auricle to the abnormal beats.

chest. During the fortnight he was under observation his pulse tracings showed remarkable variations, as in Figs. 80-83, but the interesting thing about his heart was its peculiar susceptibility to digitalis. Comparatively small doses would speedily alter the character of his pulse, producing a slow

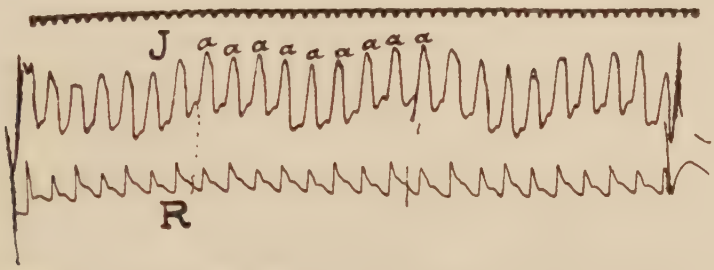


Fig. 79. Taken 3 minutes after Fig. 78. The rhythm is now normal, each radial pulse being preceded by an extra-systole. The rate is 120 per minute. The radial pulse shows pulsus alternans.

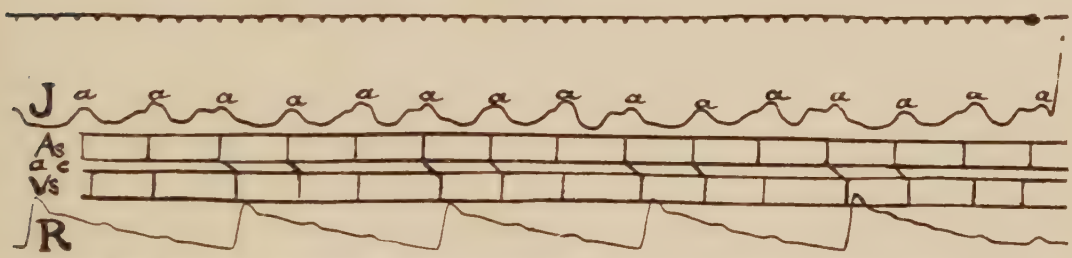


Fig. 80. Shows groups of three beats—the third in the group being an extra-systole. In the last group there are shown two extra-systoles ; the auricle is quite regular. (1920.)

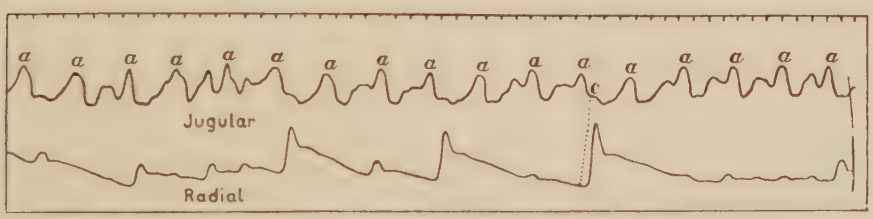


Fig. 81. Shows groups of abnormal ventricular systoles varying in number, while the auricle is quite regular. (1920.)

pulse, quite regular for long periods as in Fig. 78. In all these tracings (Figs. 76-83) it will be seen that the auricle is undisturbed when the ventricle is behaving so peculiarly.

In Fig. 83 it will be seen that there are two auricular waves to one pulse-beat. The *a* waves alternate in size, a big wave being followed by a smaller. The small wave immediately precedes the radial pulse, while the big wave falls immediately after the radial pulse. On auscultation the reason for this

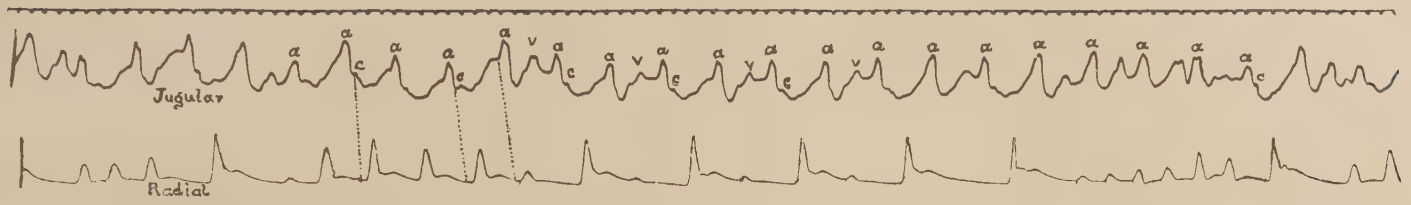


FIG. 82. Shows a very irregular radial pulse due to varying numbers of abnormal ventricular contractions. It is to be noted that these groups of small radial beats are regular in rhythm in contrast with the small beats of auricular fibrillation as in Fig. 92. (Observation 2.)

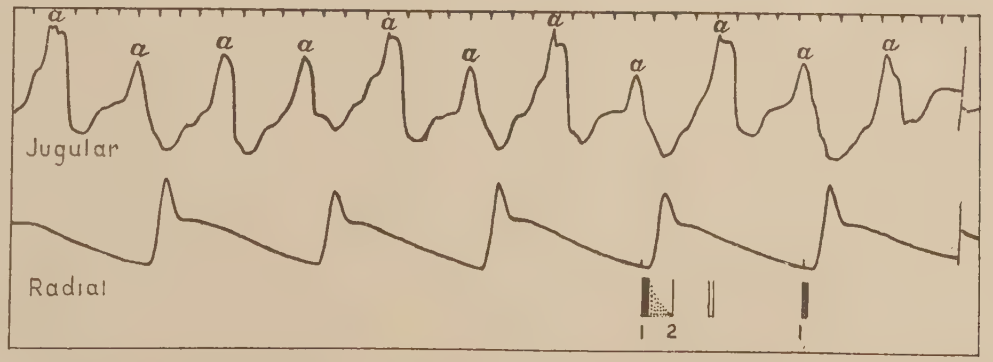


FIG. 83. Shows the effects of digitalis; the radial pulse is quite regular, the jugular is double the rate of the radial. The jugular waves alternate in size, the larger beat falling during the pause in the radial. At this time the ventricle was in systole, shown by the larger wave and by the presence of the sounds of an extra-systole. (Observation 2.)

was discovered. The first sound of the heart was followed by a murmur which ran up to the second sound, and then there was a short pause interrupted by the short sounds characteristic of an extra-systole. These sounds are represented diagrammatically under the tracing of the radial pulse. It was evident that an extra-systole of the ventricle occurred at the same time that

FIG. 83A. Record taken in 1924 from the same patient as gave the four previous tracings. When standing the rhythm was quite regular, when lying down it slowed in rate, and an extra-systole appeared after two normal beats. (Observation 2.)

the auricle contracted, so that the blood could not gain entrance into the ventricle, and was thrown back into the jugular vein, while when the ventricle was in diastole less blood was sent back into the jugular. The presence of this extra-systole lends support to the view that all the small beats in this form of paroxysmal tachycardia are of the same nature as ventricular extra-systoles.

The radial pulse shows an alternating rhythm in Fig. 83.

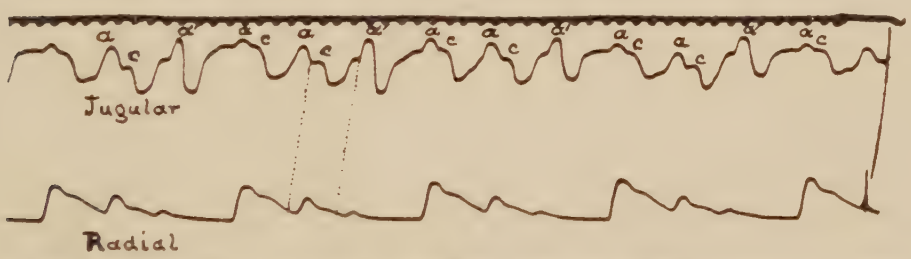


FIG. 83B. Taken 10 minutes later after Fig. 83A, showing the persistence of the extra-systole, and resembling the pulse rhythm taken four years before (Fig. 80). (Observation 2.)

The small beats.—The radial beats are sometimes absent, and when present are small and imperfect. There is a striking difference between these small beats and those in auricular fibrillation. The former are of the nature of extra-systoles, produced in the manner described (p. 139). They are, when occurring in groups, perfectly regular, although their size varies in a curious way (see Fig. 82).

But they have this peculiar resemblance to the small beats of auricular fibrillation that they may both be susceptible to digitalis, as both tend to disappear with the administration of this drug. This raises the question of

the action of digitalis, which is difficult to explain in this form of tachycardia, though some light is thrown on its action in auricular fibrillation.

Clinical diagnosis.—The patient's account of his sensations may help in the diagnosis, especially when he is conscious of the onset and offset—the latter terminating with one or two slow beats before the normal state is assumed, although by this means it cannot be differentiated from short attacks of auricular flutter.

If one bears in mind the peculiar features of the irregularity in the ventricular form, it is quite possible that with the finger on the pulse and by auscultation at the same time when there are runs of small beats interrupted by the big beat, a diagnosis may be made by the unaided senses, but I have not been able to put this suggestion to the test in a sufficient number of cases to enable me to speak with certainty.

Prognosis.—The principle which has guided prognosis in other conditions is applicable here. The abnormal conditions which provoke the disturbance and the structure which is affected by it are not in themselves of serious moment. By hampering the ventricle in the output of blood its efficiency is impaired, but if the muscle of the heart is sound, there is no danger in the condition. But where the muscle is damaged, then danger may arise, more through the damaged muscle than the abnormal rhythm. The woman from whom Figs. 69-74 were taken, and the men from whom Figs. 66, 67, 75 and 76 were taken, were only slightly inconvenienced during the attacks, which disappeared in after years. On the other hand, the two patients from whom Figs. 77-83 were taken suffered distress during the attacks, both from anginal pains and breathlessness. One was relieved by digitalis, the other died suddenly. The latter had undoubtedly a much damaged left ventricle. Observation 2 records a portion of the history of the former.

Treatment.—As the condition occurs in paroxysms, usually of short duration, there is seldom an opportunity of treating the attacks while they are present. In only one case have I found it sufficiently prolonged to enable the effects of treatment to be studied, and here digitalis was very beneficial, but I do not know whether the drug would have the same effect on other sufferers from this complaint (see Observation 2).

For the prevention of the attacks I do not know of any treatment further than attending to the patient's general health.

Rothberger gives excellent records of an attack which seems to have been stopped by quinidine (Fig. 68). The records show a condition corresponding to that represented in Figs. 66 and 67—a tachycardia arising in the genetic system in the auricle. The patient from whom Figs. 66 and 67 were taken had for weeks together frequent attacks. Some days the heart was continuously passing in and out of attacks long and short—but there was never the slowing of the rate as shown in Rothberger's case under quinidine.

OBSERVATION 2.—Male. Aged 58, seen May 26, 1920. Complains of pain over left breast, and breathlessness on slight exertion. Has lived many years in India, and has suffered from malaria and attacks of diarrhœa for several years after 1896; otherwise a healthy man.

He felt well and fit until January 1919, when, being engaged in severe bodily labour, he felt a pain in his chest which became worse until he desisted from the heavy work. At this time the weather was cold, and the pain was easily provoked when he walked out into the cold air. He went to bed for three weeks, and for a few days the pain kept recurring, but gradually abated. Since then, however, the pain can be provoked with exertion, and particularly during periods of heart irregularity, of which he is conscious. At these times he is pulled up on walking 200 or 300 yards because the pain becomes severe. The pain is more or less continuous, sometimes attaining a great severity, but never coming on in short spasmodic attacks; it is felt across the chest and down the inside of the left arm. When at his best, and when the heart is not irregular, he can walk from two to six miles on the level at a quiet pace. On an incline he is then pulled up by breathlessness, and occasionally by pain.

On examination he is a well-nourished, healthy-looking man, though slightly sallow; heart enlarged, dullness extending one inch beyond left nipple line; a systolic murmur loudest at the apex; rhythm markedly irregular. A slight trace of albumin in the urine; an X-ray examination showed nothing but a slight enlargement of the heart.

A tracing was taken of his radial and jugular pulses; it showed a very disorderly rhythm varying in a peculiar manner, of which Figs. 80-83 are examples. It is evident that the irregularity is due to ventricular beats independent of the auricular beats, except at the beginning of a group, where there is a large radial beat, which is seen to be preceded by an auricular wave.

Hitherto the cases of this condition I had seen were paroxysmal, and I had no clear idea of the treatment. Finding this arrhythmia was not paroxysmal but persistent, we put him upon digitalis (1 drachm per day) on May 29. That night he was very ill, suffering from an attack of cardiac asthma and Cheyne-Stokes breathing, with pain across the chest. He speedily responded to the treatment, and the second day his pulse became at times slow and regular, as in Fig. 83, and at other times irregular, Fig. 82. As soon as his pulse became slow he felt remarkably well; pain disappeared, and he was able to take a considerable amount of effort without distress. The digitalis was stopped to see whether the improvement was due to the drug, and in a few days he was as bad as ever, the small beats tending to recur, as shown in Fig. 81. He was again put on digitalis, and in a couple of days he was greatly improved; the pulse became slower, and many normal beats appeared. He was told to watch the effect of the drug, and to diminish the quantity when he felt very well, and take it just when he began to feel the slightest return of his weakness, so that he should regulate the quantity of his drug according to his condition. He continued well during the ten days he was under observation, and seven months later he wrote saying he was still very well, and continuing to take digitalis.

A year later I saw him again. He was free from any distress, and small doses of digitalis were sufficient to prevent the heart running away.

I saw him again on July 18, 1924. He was looking well, and was able to walk several miles without distress. He takes digitalis occasionally, and thinks it does him good, but on inquiry it is but an occasional dose, and it is evident that the long attacks no longer occur. He had taken no digitalis for two weeks when I saw him. When he stood his pulse was quite regular; on lying down it became irregular in a few minutes, as in Fig. 83. The irregularity persisted as long as he lay, as in Fig. 83. As soon as he stood up and walked about, the rate increased to nearly 100, and became regular.

CHAPTER XXVII

Auricular Fibrillation
(Synonyms: Paralysis of the Auricle, Nodal Rhythm)

Historical account.
The term auricular fibrillation.

Historical account.—In the history of medical progress it is of interest to note that, while some discoveries are recognized at once and an exalted view taken of their possibilities, which a fuller experience may or may not endorse, other discoveries are not recognized or appreciated till long years after, when experience gradually reveals their significance. The condition which at one time has passed under such terms as paralysis of the auricle, and nodal rhythm, and is now called auricular fibrillation, belongs to the latter group. When the full significance of the discovery of this condition comes to be recognized, it will be looked upon as one of the most important discoveries, not only in cardiology but in medicine; while its pursuit continues to throw a new light upon many clinical and physiological problems. Already its study has revealed principles, hitherto unsuspected, which govern vital activities which are applicable to every field of medicine. Some of these are as yet only dimly perceived, and it needs but time and experience for them to be clearly recognized.

A brief account of the history of this condition serves to illustrate how research in clinical medicine reveals the nature and function of organs which are beyond the reach of physiological or other experimental methods. For more than one hundred years clinical observers were dimly conscious of a peculiar condition of the heart which produced ill-health in certain people. The description by Withering, of the effect of digitalis, shows that he recognized a form of heart failure which was peculiarly susceptible to the drug. After his time, when physicians recognized mitral stenosis, they came to associate a peculiar pulse which frequently was present in this condition, called the ' mitral pulse '. As long as forty or fifty years ago, some physicians recognized that in mitral stenosis the presystolic murmur sometimes disappeared.

In carrying out an inquiry into irregularities of the pulse, I recognized that the radial pulse gave information only of what the left ventricle was doing, and I endeavoured to find out what the auricles were doing. Over thirty years ago I recognized that in certain conditions the auricle gave no

evidence of activity, but it was not till twenty-six years ago I discovered that in certain patients all evidences of auricular activity suddenly disappeared.

OBSERVATION 3.—The first patient in whom I clearly recognized this, was a woman who had been under my care for seventeen years. She had shown for some years a presystolic murmur at both the mitral and tricuspid orifices. She had also shown pulsation in the jugular veins, and in the liver, due to the auricular systole (Figs. 84, 85, 86). Suddenly, extreme heart failure set in, the presystolic murmurs disappeared as well as the auricular waves from the jugular and liver tracings, and the heart became irregular. Following the custom of the day (Fig. 86), I assumed that with the disappearance of all signs of auricular activity the auricle had become paralysed.

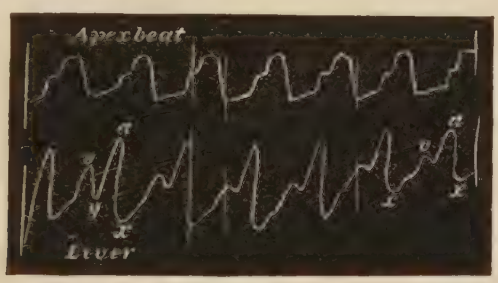


FIG. 84. The liver pulse shows a well-marked wave (*a*) due to the auricle. (Observation 3, 1892.)

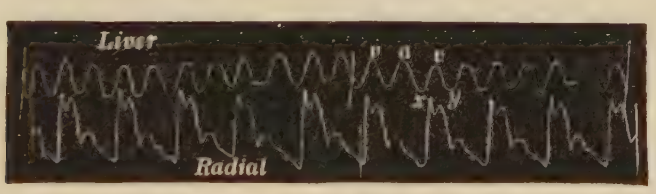


FIG. 85. There is still a well-marked wave in the auricle. (Observation 3, 1897.)

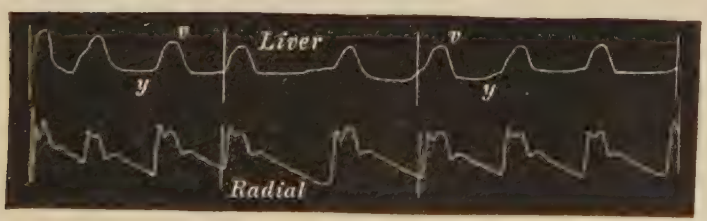


FIG. 86. Showing the irregular rhythm, characteristic of auricular fibrillation. When compared with Figs. 84 and 85, it will be seen that there is no auricular wave in the liver pulse, and the heart's action is irregular. (Observation 3, 1898.)

At this date little was known of the processes concerned in regulating the heart-beat. The heart-beat was supposed to start somewhere in the auricle, and I assumed that as the auricle ceased to beat, the ventricle started on a rhythm of its own. When later, the auriculo-ventricular node and bundle

were discovered, and it was shown that the ventricular contraction was dependent upon the a.-v. node, I suggested that the contraction started here, and called the condition ' Nodal Rhythm '.

In 1906, Cushny and Edmunds drew attention to the similarity of the irregularity of the pulse of a dog in whom they had produced auricular fibrillation, to that of a patient with an irregularly acting heart, and they suggested that auricular fibrillation was the cause of the patient's abnormal rhythm. This view was confirmed by Winterberg and Rothberger and Lewis in 1909. The demonstration seemed so convincing that, although ignorant of its nature, I, with others, unfortunately accepted the discovery as settling the problem.

This conception of auricular fibrillation as the cause, started investigations upon a new line which one must confess has been of little profit. In reading the recent literature upon this subject it is astonishing to find how obsessed clinical observers are by laboratory discoveries. A number of writers reckon to give the history of this discovery, and they start with the time when it was labelled auricular fibrillation, evidently unaware that all the signs by which it is recognized, the basis of prognosis, and the indications for treatment, were all recognized before the term auricular fibrillation was applied to it. As a matter of fact, the recognition that the auricle was fibrillating has not added one single fact to the clinical aspect of the subject.

As years went on I became convinced that there was a great deal more in the matter than a fibrillating auricle. I had for many years recognized that with the onset of this condition the heart acted in many ways in a manner different from that when the normal rhythm was present. The character of the pulse became quite distinctive. The rate of the pulse could often be influenced by effort, rest, and digitalis, in a manner quite different from the normally acting heart. The most striking of all was the distinctive form of heart failure that frequently set in, sometimes immediately on the onset of this abnormal heart action. Moreover, patients who might be suffering from all the symptoms of heart failure which is characterized by angina pectoris would suddenly lose all the symptoms of angina, and would suffer from the symptoms peculiar to auricular fibrillation.

The term auricular fibrillation.—It is one of the misfortunes of medicine that names are given to phenomena before their nature is understood. The consequence is that such terms are accepted as explanations, and such explanations are invariably wrong. This is what happened when the term fibrillation was applied to this condition. There is no doubt the auricle is fibrillating, but the impression is formed that the fibrillation constitutes the disease, whereas it is but one of the effects. Consequently a great deal of time and energy has been spent uselessly in searching for the nature of the fibrillating auricle in the auricle itself. One might as well examine a dog's tail to find out why it wags. It turns out that the terms originally employed, ' paralysis of the auricle ' and the ' nodal rhythm ', are nearer the truth, although I now recognize that I had an imperfect conception of what these terms implied. In using the term nodal rhythm I imagined that the auricle being paralysed,

the a.-v. node took on the function, and started the ventricular contraction, just as the bundle takes on these functions in complete heart-block. The further inquiry into the subject shows that this is a true interpretation, for it will be found that the due appreciation of what happens when the auricle passes into fibrillation can only be understood when the a.-v. node is looked upon as an organ as distinctive as the s.-a. node.

The employment of the term paralysis has also been extremely suggestive. The view that auricular fibrillation is due to the loss of control of the s.-a. node leads to a better conception of what is meant by paralysis, even when employed in the description of diseases of the nervous system. It is a remarkable fact that physiologists and neurologists have paid very little attention to organs when their nerve supply has been cut off. It must be remembered that these organs are still supplied with blood, that their cells are still living, and therefore like all living cells are capable of receiving impulses and discharging their energy, and when they do this they are capable of giving rise to a variety of phenomena. When this happens to the heart, disturbances of a particular kind arise which it was impossible to interpret until the function of the genetic system and the part it plays in the normal and abnormal heart was understood.

It has already been pointed out that the s.-a. node controls and regulates not only the auricle but also the a.-v. node, and through it the ventricle. Consequently when it ceases to exercise its functions the loss of control affects not only the auricle but the a.-v. node, and while the fibrillation of the muscle of the auricle is evidence of the loss of control, it must be remembered that this gives no information of what happens to the a.-v. node when the control is lost. In the following descriptions of auricular fibrillation and flutter it will be found that it is the uncontrolled a.-v. node which requires particular attention.

One should be chary of inventing new names or of altering old ones, for the name usually represents the imperfect knowledge at the moment it is given. While I am of opinion that 'nodal rhythm' is a more accurate term than auricular fibrillation, as it is the behaviour of the a.-v. node that gives rise to the peculiar action of the ventricle, and it is on this structure that the sympathetic and vagus act, yet as the matter is not finally settled, I retain the term auricular fibrillation as it is now in common use. It will be time enough to change the name when more is known of the condition.

CHAPTER XXVIII

The Diagnosis of Auricular Fibrillation

Appearance of the patient.
The distress in breathing.
The pulse.
The sounds.
The jugular pulse.
Graphic records.
The radial pulse.
The jugular pulse.
Fibrillation waves.
The electro-cardiogram.

Appearance of the patient.—In many patients one suspects the presence of auricular fibrillation at the first glance. A dusky face, with blue lips and blue ears, laboured respiration on moderate effort suggests its presence, while in the more advanced conditions the propped-up position assumed in bed, and the distress in breathing produced by slight movements, are still more confirmatory. The reason for assuming the possible presence of this condition is that they are evidences of the diminution of output of blood from the heart, and the most common cause of this diminished output is the small imperfect and ineffective beats which occur in auricular fibrillation. Other morbid conditions of the heart give rise to the same symptoms, but they are not so common.

The distress in breathing.—The patient's description of his limitation is helpful, especially when he describes the distress which limits his power to respond to effort. This is usually distress in breathing. The onset of the distress is often so accurately described and so characteristic that the diagnosis can be made from this alone. The story usually is, that while making an effort such as going rapidly up a hill or running, or lifting a weight, the patient became conscious of his heart running away, or he suddenly became very breathless. Every case of ' heart failure ' with respiratory distress from what is called ' heart strain ' which followed some extra effort that I have seen could be attributed to the onset of auricular fibrillation or its allied condition flutter.

When there is not this distinctive onset there is a history of a gradual increase of breathlessness. The fibrillation has come on but the patient is unaware of the change and continues to live his ordinary life. Slowly and

imperceptibly the limitation creeps on, the first signs of which are brushed aside and attributed to ' being out of condition '. By and by the encroachment upon the field of response becomes so marked that the patient consults his doctor.

On the other hand, there may be no such limitation, and the individual carries on not a bit the worse for the onset of fibrillation. In these cases the rate is not increased, and the discovery of the peculiar irregularity is made during a routine examination for some other purpose, as life insurance.

The pulse.—The physical examination begins with the pulse, which is found irregular, being of that disorderly kind shown in such tracings as

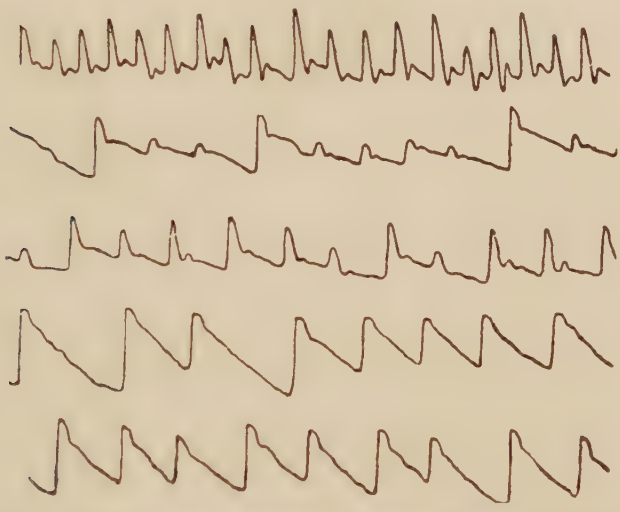


FIG. 87. Tracings taken from different patients showing different forms of the pulse in auricular fibrillation.

Fig. 87. Sometimes it may not be easy to make this out by the finger, then the ear, which is much more sensitive to variations of time, may give the required information. There are often present numerous small beats which the untrained finger may fail to detect. This is sometimes spoken of as a ' pulse deficit '.

The sounds.—When the sounds are free from murmur, or when there are systolic murmurs present, the irregularity is the distinctive feature found on auscultation, but when there is mitral stenosis the sounds are practically diagnostic in the majority of cases.

This is a point that I would like to lay stress upon, and urge the doctor to familiarize himself with the sounds of the heart so that he can time the murmurs. It came as a revelation to me that I could get few doctors, even

highly trained physicians and professors of medicine, to appreciate the change in the sounds that took place after the onset of auricular fibrillation. I have mentioned that the physicians forty or fifty years ago had noted that the presystolic murmur sometimes disappeared. They could not tell the reason for the disappearance, and they only recognized it when the heart-rate was slow. I have repeatedly seen physicians describe the diastolic murmur of mitral stenosis in a slow acting heart as being due to aortic regurgitation. The possibility of it being mitral in origin had never dawned upon them. When the rate of the heart is increased this diastolic murmur may fill the whole period of diastole, and it is then assumed to be mitral in origin and put down as a presystolic murmur, meaning thereby a murmur produced by the systole of the auricle. In nearly every case there are occasional pauses between the beats much longer than usual, and when this happens the murmur follows the second sound and fades away, and there is a short silence before the first sound. If a doctor would put out of his head his preconceived ideas about the presystolic and diastolic murmurs, and merely note the relation of the murmur to the sounds, he would have no difficulty in recognizing that the murmur in auricular fibrillation is diastolic in time, being always related to the second sound, and having a variable relation to the first sound during the irregularity of the heart's action. This is diagrammatically shown in Figs. 88 and 89.

I am insistent upon this point, for I found at one time it was impossible to get clinical observers to recognize these facts. On one occasion I was demonstrating a patient where the pulse-rate was about 120 beats per minute, to four physicians, three of them professors of clinical medicine. I tried to get them to listen particularly during the long pauses which occasionally occurred, when there was a silence before the first sound, but they were all agreed the murmur was presystolic in time, and would on no account accept my description. One of them watched the case with me for five days, during which time under digitalis the rate decreased to between 60 and 70 beats per minute. There was only then heard a murmur following immediately the second sound tapering away into silence before the first sound, a change which surprised the physician, who said he would have been disposed to consider the murmur aortic in origin. I pointed out to him the area in which the murmur was heard was diagnostic, being limited to the apical region of the heart. During twelve years I repeatedly called attention in articles and in my book on the pulse to the characters of the murmur in mitral stenosis before and after the onset of auricular fibrillation ; few, if any, recognized the alteration till Lewis demonstrated it by means of the electro-phonograph.

This experience has led me to recognize that a great many people never acquire the ability to time murmurs, and I have advised doctors not to listen to hearts perfunctorily or as a mere matter of routine, but in every case to listen intelligently to the differences in the sounds and to familiarize themselves with timing the sounds with the pulse, the apex beat, or the movements in the neck.

I urge this all the more earnestly, for the reason that the whole tendency of medicine is to employ devices which do not require the education and refinement of our senses for observation. It must be remembered by teachers

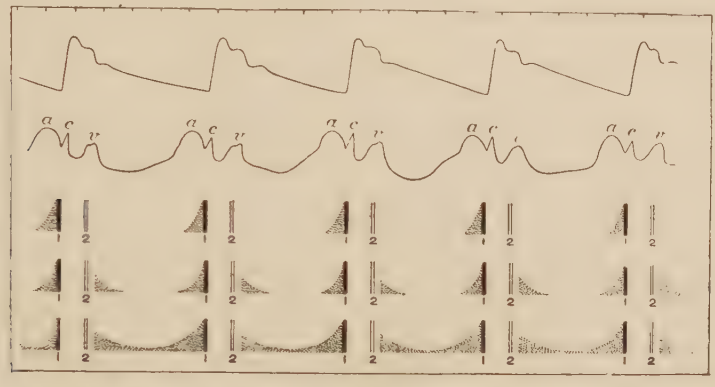


FIG. 88. Tracings of the radial and jugular pulse with diagrammatic representation of the changes in the murmurs of mitral stenosis. The downstrokes 1 and 2 represent the first and second sounds of the heart. The first indication of stenosis is the appearance of the murmur before the first sound, and on reference to the jugular tracing this murmur will be found to coincide in time with the wave *a*, due to the systole of the auricle. With an increase in the stenosis, a short diastolic murmur appears, immediately after the second sound, and this is represented in the middle diagram. A further increase in the stenosis, shown in the lowest diagram, leads to the prolongation of the diastolic murmur, which in combination with the presystolic murmur fills up the whole interval between the second and first sounds. (For the next change see Fig. 89.)

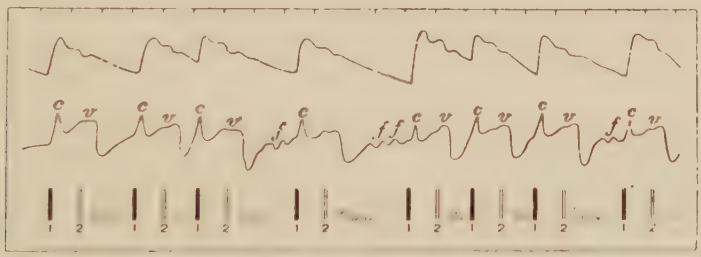


FIG. 89. After the changes described in Fig. 88, the next change is a sudden alteration of the rhythm of the heart. In Fig. 88 the heart is quite regular. In Fig. 89 it is irregular. The wave *a*, which is a marked feature in the jugular tracing of Fig. 88, is absent in Fig. 89. The presystolic portion of the murmur is also absent in Fig. 89, this feature being best appreciated during the longer pauses of the irregularity. The disappearance of these evidences of auricular activity and the irregular action of the heart indicate the onset of auricular fibrillation. The waves *ff* are due to the fibrillating auricle.

that the vast majority of doctors have not the opportunities for either graphic or electro-cardiographic instruments to help them, and that their own powers of observation are infinitely superior to all these methods, for the doctor

has not only to recognize but to assess the value of the symptoms and signs which he detects.

The jugular pulse.—Another distinctive sign which the doctor may observe in his patient is the time of the jugular pulse, which is synchronous with the radial pulse (Fig. 90). But to distinguish the auricular form of

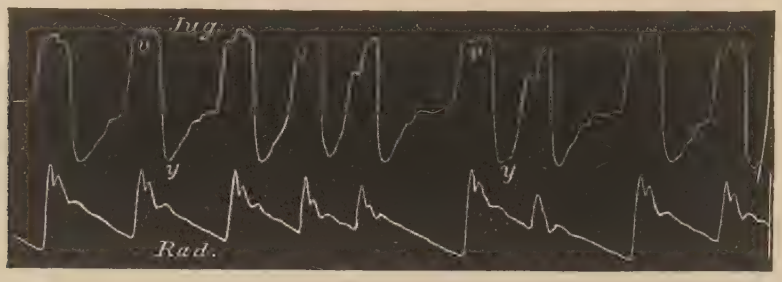


Fig. 90. Simultaneous tracings of the jugular and radial pulses, showing the ventricular jugular pulse, *i.e.* one large wave (*v*) synchronous with and due to the ventricular systole, and one large fall (*y*) synchronous with and due to the ventricular diastole. The rhythm of the heart is continuously irregular. When there are long pauses the tracing of the jugular gradually rises before the next large wave, on account of stasis in the veins. When the pause is short there is no sign of stasis, or only a slight wave, as on the second beat, where it might simulate a wave due to the auricle, but the real cause is seen to be stasis in the veins when the pause is longer.

venous pulse which is indicative of the normally acting heart from the ventricular form of venous pulse which is indicative of auricular fibrillation requires a good deal of patient observation and training. Nevertheless, there is no reason why an observant doctor should not acquire the power (see Chapter III.).

Graphic Records

The radial pulse.—The records of the radial pulse show a continuous irregularity where the pauses between the beats are ever varying in duration, while the sizes of the beats are also varying. There is no order of any kind—it is a disorderly irregularity of a kind that occurs only in auricular fibrillation and to a less degree in flutter.

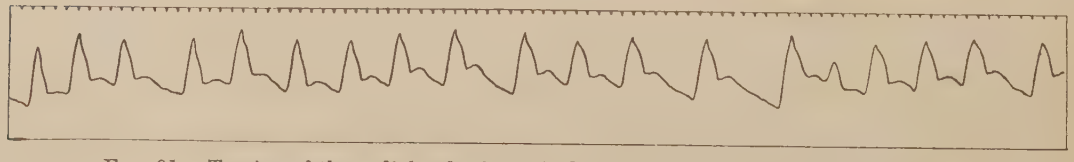


Fig. 91. Tracing of the radial pulse in auricular fibrillation where the size of the beat varies with the length of the preceding pause. (Observation 5.)

It is possible to distinguish two kinds of pulse-beats. In one the beats are fairly full and complete, and vary in size according to the length of the preceding pause, a large beat following a long pause, and a small beat following a short pause (Fig. 91). In the second form there is not this relationship.

The beats are manifestly imperfect and often ineffective, the size of the beat having no relation to the length of the preceding pause—a large beat following a short pause, while a small beat may follow after a long pause (Fig. 92).

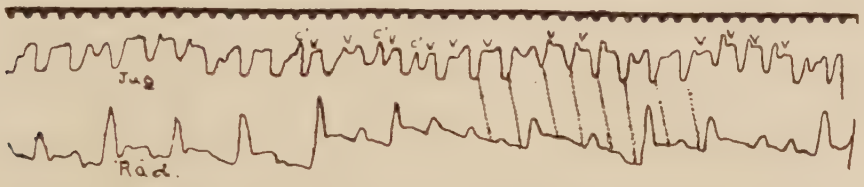


Fig. 92. Tracings from a patient with auricular fibrillation where the size of the beats do not vary with the length of the preceding pause. (Observation 11.)

This difference in the character of the pulse-beats has hitherto escaped recognition. The different forms of pulses have a profound significance, and their investigation has led to the explanation of the nature of heart failure, besides throwing a flood of light upon a number of phenomena. Some of these will be dealt with later.

The jugular pulse.—In Chapter LXVIII. different forms of the venous pulse are discussed. It is sufficient here to point out that in a normal venous pulse there is always a wave due to the auricle which precedes the carotid wave *c* (Fig. 3). With the onset of fibrillation of the auricle the auricular wave disappears and there is instead a large wave due to and synchronous with the ventricular systole in Figs. 90 and 92. In some patients with slow-acting hearts we can see the large jugular waves passing up the neck with each contraction of the ventricles. Fig. 90 is an example.

OBSERVATION 4.—The change in the character of the venous pulse is sometimes very striking. The patient from whom Figs. 93 and 94 were taken had mitral and tricuspid stenosis, and large waves were at one time visible in the jugular, due to the auricular systole as in Fig. 93.

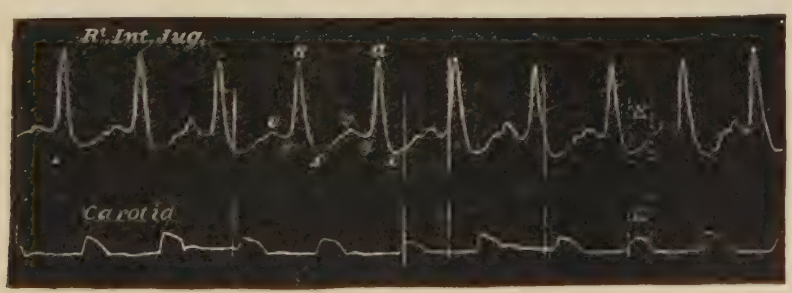


Fig. 93. Simultaneous tracings of jugular and carotid pulses showing a large wave (*a*) due to the auricle. Compare with Fig. 94. (Observation 4.)

The patient was conscious of these waves, and one morning she said to me that the beating had gone from her neck. I examined her and took a tracing from the usual place in the neck and obtained Fig. 94, which shows the disappearance of the auricular waves and the appearance of small broad waves, ventricular systolic in time. The heart had become irregular, and the mitral and tricuspid presystolic murmurs had disappeared.

Fibrillation waves.—Although the auricle is in fibrillation the fibres may contract in a peculiar rhythmical manner which is reflected by the blood into the jugular veins, so that there is sometimes found a series of rapid waves varying in size and in rate. These movements are not constant, sometimes

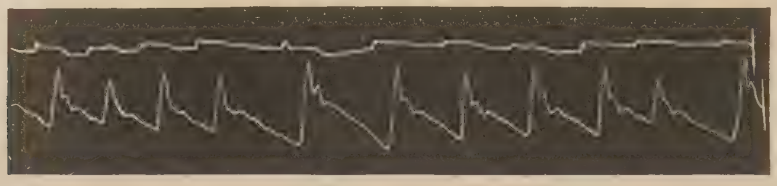


Fig. 94. Simultaneous tracings of a slight movement in the neck and of the radial pulse. The rhythm is now continuously irregular, with a tendency to long pauses. The tracing from the neck was taken from the same situation which gave the jugular pulse in the preceding figure, and demonstrates the absence of any wave due to the auricle. (Observation 4.)

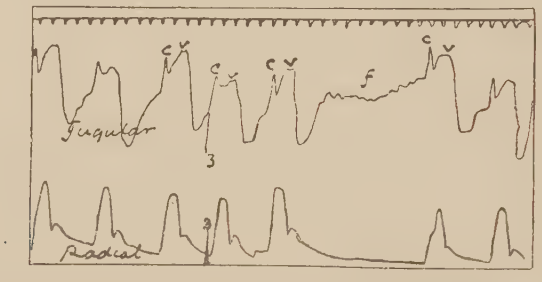


Fig. 95. A tracing from a patient with auricular fibrillation, showing small fibrillary waves (*f*) in the jugular tracing.

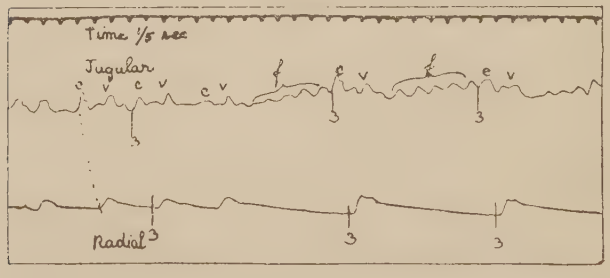


Fig. 96. The jugular tracing shows coarser fibrillary waves (*f*).

being well marked in one part of the tracing and absent in the other. They only appear during some of the long pauses between the ventricular contractions (Figs. 95, 96, 97, and 106).

The electro-cardiogram.—The electro-cardiograph record shows the normal ventricular complex with an absence of the auricular wave *P*. The rhythm is invariably irregular. Between the ventricular complexes the record shows

frequently irregular movements, and occasionally movements fairly regular at a rapid rate (Fig. 98). It is likely that the origin of these movements and the small waves of the veins are due to numbers of the fibres contracting more or less regularly, as they resemble the somewhat regular movements to be found occasionally in fibrillation of voluntary muscles.

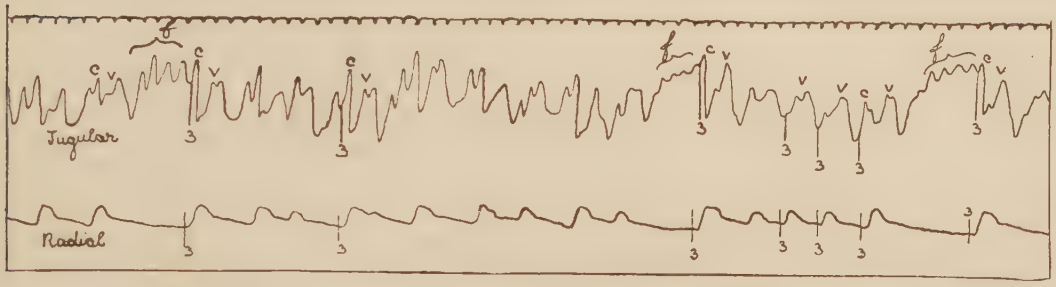


FIG. 97. The jugular tracing shows fibrillary waves (*f*) of different sizes.

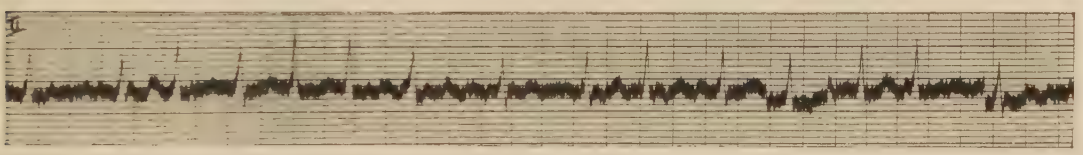


FIG. 98. An electro-cardiogram of auricular fibrillation showing the absence of the *P* waves and the irregularity characteristic of auricular fibrillation.

There is a tendency to regard them as due to a 'circus' movement, but as no one seems to understand the factors concerned in a circus movement, the statement is not very illuminating. As they resemble the fibrillary movements of voluntary muscle, both have possibly the same origin. This is one of those matters which require further investigation.

CHAPTER XXIX

Cases illustrative of Auricular Fibrillation

Observations 5-17.

General descriptions alone are usually so vague and indefinite that there is a difficulty in applying them to individual cases. Even when individual cases are recorded, and when numerous devices are employed to find out the symptoms, the latter are frequently so unsystematically recorded that the essential features are lost in a mass of detail.

I have long recognized the difficulty of describing this condition in a manner that will enable the general practitioner to recognize the phenomena and so establish a diagnosis and provide a prognosis. I have therefore attempted to give this kind of information, and in quoting records of cases to limit the description to essential matters. With this idea in my mind I give records here of a number of cases typical of the various aspects as found in general practice.

During the past twenty-five years I have made a close study of patients with auricular fibrillation and have records of several thousands. I have endeavoured to obtain a means of diagnosis by my unaided senses. This was necessary, because a diagnosis had often to be made under circumstances which precluded the use of instrumental devices.

OBSERVATION 5.—In 1907 I was consulted by a man 60 years of age, who had been under treatment for some months on account of his heart. For ten years he had been rather short of breath, and his heart ' fluttered ' at times. On examination the heart showed no abnormality beyond a well-marked irregularity characteristic of auricular fibrillation (Fig. 91). The rate was between 50 and 60 beats per minute, and was not markedly increased on effort. There was an absence of small beats. Since that time he has led an active business life. I have seen him from time to time, and the auricular fibrillation was present on every occasion, shown by pulse records and the electro-cardiograph. Beyond being somewhat circumscribed in his effort on account of breathlessness, he has not suffered from any form of heart failure, and at the age of 77 continues to lead a fairly active life.

OBSERVATION 6.—Male. This patient first consulted me on November 25, 1900, his age being then 51, complaining of shortness of breath on exertion. He had felt well until the previous year, when he had attacks of shortness of breath and a choking sensation which passed off. During the last few months the shortness of breath had come on with the slightest exertion, and his legs had begun to swell.

There was no history of rheumatism or other infectious complaint.

He was a healthy-looking, muscular man. The pulse was continuously irregular, and tracings taken at this time showed the irregularity to be characteristic of auricular fibrillation. The

heart was slightly enlarged, the dullness extending to the nipple line. He was put to bed for a few weeks and given digitalis, and he improved remarkably. I did not see him after September 2, 1901, until March 20, 1905. In the meantime he had worked out a method of treating himself by making a decoction of foxglove leaves. When his legs swelled so that he could not lace his boots comfortably, he found he was short of breath, and he took his decoction for a few days till the swelling disappeared and his breathing became easier. On this date I noted that the heart had not altered, the pulse was still irregular, and the jugular veins were very full. From this date I saw him at frequent intervals till 1909. My last examination of him was on January 22, 1909, when he was on his second honeymoon. He looked well, save for a slight yellow tinge in his face. The heart's dullness extended one inch beyond the left nipple line. There were no murmurs. The heart was still irregular, and an electro-cardiogram showed the characteristic signs of auricular fibrillation. I heard from him on March 26, 1913, when he wrote saying he was getting along very well: 'The more I walk out the less dropsy I have in my legs. I still keep my foxglove leaf by, and I am positive I could not live without it.' In 1920, when last I heard of him, he was still in fair health though feeble (aged 71).

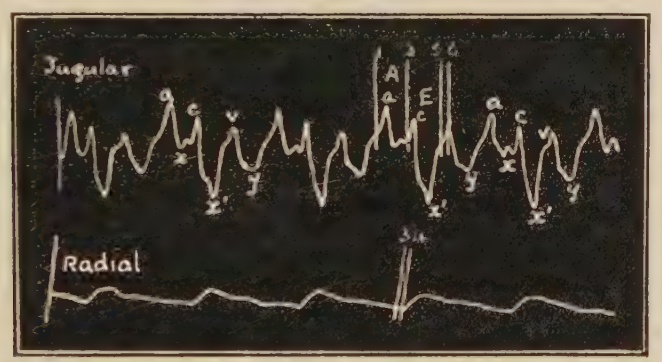


FIG. 99. Shows a regular rhythm and an auricular venous pulse and wide *a-c* interval (space *A*). (Observation 7.)

FIG. 100. Shows a regular rhythm and an auricular venous pulse and wide *a-c* interval (space *A*). (Observation 7.)

OBSERVATION 7.—Male. Born 1851. I attended him for an attack of rheumatic fever in 1883. He was left with a damaged mitral valve, shown by a systolic murmur, but remained in fair health till 1897, when he had serious heart failure with dropsy. From this also he made a good recovery. I had taken tracings of his apex, radial pulse, and jugular pulse at frequent intervals after 1892. His heart was invariably regular except for a short period in 1897, the irregularity being due to dropping out of ventricular systoles (partial heart-block, Fig. 172). His jugular pulse was always of the auricular form, a peculiarity in this case being a persistent increase in the *a-c* interval. Tracings of the jugular and radial pulses, taken in 1892 and 1903, are given in Figs. 99 and 100. The rhythm in these records is regular, and the auricular wave *a* is

well marked in the jugular tracings. A presystolic murmur was noted in 1892, and afterwards there gradually developed a diastolic murmur. In 1903 there was a long diminuendo murmur after the second sound and a well-marked presystolic murmur, the loudest portion being separated by a brief interval from the first sound (shading in Fig. 100). In the numerous tracings of the apex beat taken up to April 12, 1904, there was always a well-marked auricular wave, *a*, preceding the large wave of the ventricle, as in Fig. 102. When he visited me on the last-named date I found his heart continuously irregular, the rate between 40 and 50, and on taking tracings of the jugular pulse I found it of the ventricular type (Fig. 101); the presystolic murmur had

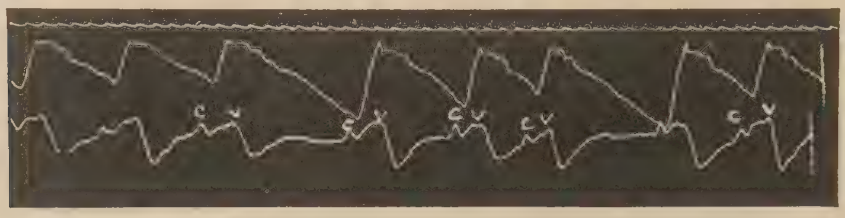


Fig. 101. Shows an irregular rhythm with the absence of the auricular wave from the jugular tracing. (Observation 7, April 12, 1904.)

gone, and there was at the time only a diastolic murmur. The wave due to the auricular systole had also disappeared from the apex tracing. That is to say, with the appearance of the ventricular venous pulse and continuous irregularity, all evidences of the contraction of the right and left auricles had disappeared. When this patient called to see me in the following week his heart was perfectly regular, the auricular wave present in the venous pulse, the presystolic murmur and the auricular wave in the apex tracing also present (Fig. 102). These conditions

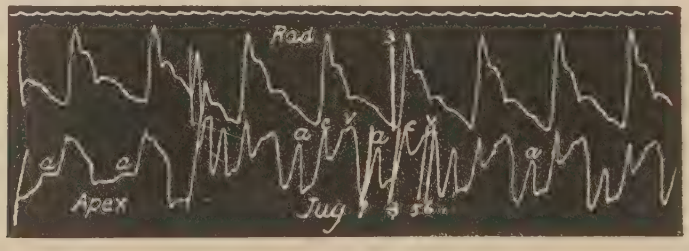


Fig. 102. Simultaneous tracings of the radial and apex, and of the radial and jugular, taken a fortnight after Fig. 101, showing a regular rhythm and the evidences of auricular contraction (*a*) of the apex and jugular tracing. (Observation 7, April 26, 1904.)

continued until November 1904, when his heart again became irregular, and all evidence of auricular systole disappeared (Fig. 103). From that date up to 1913 the heart remained in this state, and on the numerous occasions when I have taken long tracings I have never yet found the heart regular. Electro-cardiograms taken in 1910 showed the features characteristic of auricular fibrillation.

In 1904, when the auricular fibrillation occurred in this patient, he was working at his trade of a mechanic. His work consisted mainly in swinging a fore-hammer. He was quite unconscious of the onset of auricular fibrillation, and was in no way restricted by its presence. He continued to work at his trade till 1913, when breathlessness set in and dropsy, and he died a year later.

This patient, although able to follow his work, admitted that for a long time he was rather breathless on going up hills. While he had always a pulse between 50 and 60 with the normal

rhythm, I frequently found it as slow as 40 (Fig. 103), after the onset of auricular fibrillation, and it was not markedly increased on effort.

OBSERVATION 8.—A man (37 years of age) who suffered from rheumatic fever in his boyhood and had led an active life as a gamekeeper was seized on two occasions with attacks of great prostration and breathlessness. These attacks lasted some hours, then ceased suddenly, with immediate relief. Three days before I saw him he had another attack which had not ceased. He was breathless, and had to sit upright in bed, breathing in a laboured fashion. His

FIG. 103. Shows a slow irregular rhythm with the ventricular form of the venous pulse. (Observation 7.)

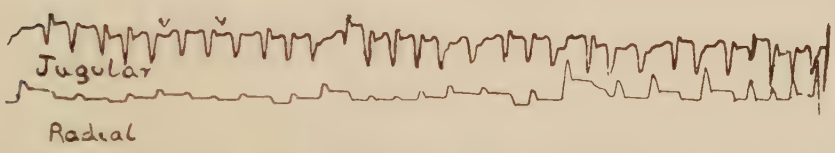


FIG. 104. Tracing of the jugular and radial pulses three days after the onset of abnormal heart action and great heart failure. The radial pulse was scarcely perceptible to the finger, and the tracing was got with difficulty. The jugular pulse is of the ventricular type, each wave corresponding to a radial pulse beat. The rate is 170 beats per minute. (Observation 8.)

FIG. 105. Tracing of the radial pulse from the same patient as Fig. 104, showing the effect of digitalis. The rate is 65, and the beats are now full and effective, and all signs of heart failure have disappeared. (Observation 8.)

face was dusky, and his lips and ears slightly cyanosed. The pulse was small and rapid, and a tracing was taken with difficulty. The rate was 160 per minute, consisting mostly of imperfect beats (Fig. 104). The heart was slightly increased in size. After a couple of days he was given digitalis, and three days later the rate gradually began to fall, and on the sixth day after beginning with the digitalis it fell to about 60 beats per minute, with a disappearance of the imperfect beats (Fig. 105). The digitalis was stopped and resumed when the rate began to increase, until the dose was found which kept the rate at about 60 beats per minute. With the fall of rate all

signs of distress disappeared, and at the end of three weeks he was able to resume his work, and experienced no discomfort so long as he took digitalis.

OBSERVATION 9.—A man of 28 consulted me for weakness and great breathlessness on exertion. When lying down he felt quite comfortable. The heart was slightly enlarged, and there was a diastolic aortic murmur and the rate was irregular, characteristic of auricular fibrillation. The rate after resting for some time fell to 50 beats per minute (Fig. 106). On getting up and walking up one flight of stairs and back, the rate increased to 170 (Fig. 107) beats per minute, and he became very breathless. Under digitalis this tendency to an increased rate lessened, so that after four or five days the rate increased to between 70 and 80 beats per

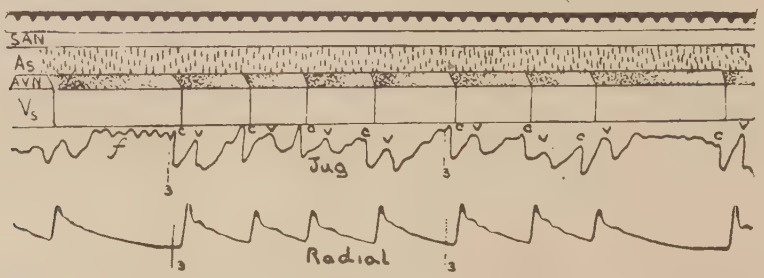


FIG. 106. Simultaneous tracings of the jugular and radial pulse when at rest. The diagram shows sino-auricular node inactive, the auricle in fibrillation, the auriculo-ventricular node varying in activity, giving rise to the peculiar radial pulse. All the radial beats are full and effective. Compare with Fig. 107. Note the fibrillation (*f*) waves in the jugular pulse. (Observation 9.)

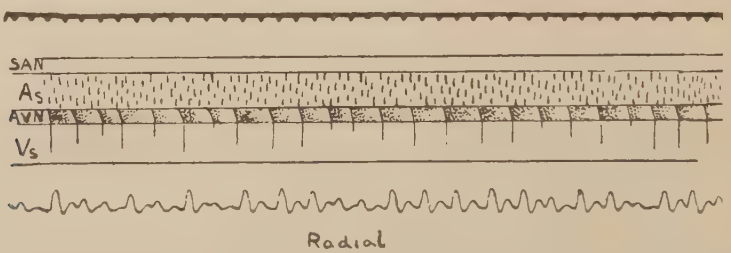


FIG. 107. Tracing of the radial pulse taken from the same patient as Fig. 106 after slight exercise. The rate is 170, and the pulse beats are imperfect. The nature of the condition is shown by the diagram. (Observation 9.)

minute after the same amount of effort. Four or five days after stopping digitalis the rate again became greatly increased on slight effort. Digitalis was resumed, and a quantity taken which kept the rate within moderate limits on effort, and he felt much better.

OBSERVATION 10.—A female consulted me in 1895. She gave a history of rheumatic fever in her youth. The response to effort was fair. The heart was not enlarged, but she had a presystolic murmur, and the rhythm was quite regular (Fig. 108). In 1896 (age then 32) I attended her during her confinement. In the course of the next four years she developed in addition a diastolic murmur, so that ultimately the whole diastolic period of the heart was filled by these two murmurs. In 1900 she became ill with breathlessness; her face became dusky and her lips blue. There was slight dropsy and enlargement of the liver. The heart's dullness extended beyond the left nipple line. The pulse was irregular, and the jugular pulse had changed from the auricular to the ventricular type. There was a long diastolic murmur, the presystolic had disappeared (see Figs. 109, 110, 111). With rest in bed and digitalis she gradually improved. As years went on the digitalis lost effect, extensive dropsy set in, with

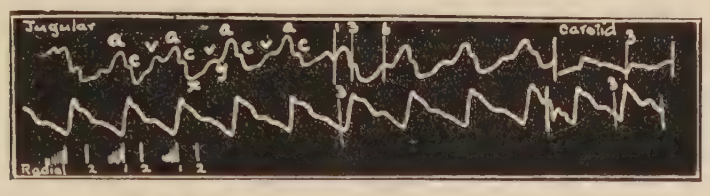


FIG. 108. Simultaneous tracings of the jugular and radial pulses in the first part, of the carotid and radial in the latter part. The jugular tracing shows the form characteristic of the auricular venous pulse where the wave *a*, due to the auricle, precedes the carotid wave *c* (downstroke 3). The shading underneath represents the time of the presystolic murmur. (Observation 10, November 5, 1895.)

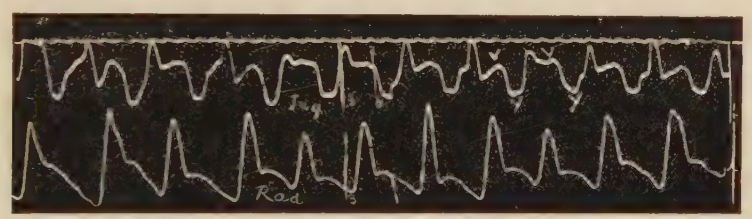


FIG. 109. The jugular pulse is now of the ventricular form—no auricular wave precedes the downstroke 3, and the rhythm is irregular. (Observation 10, March 19, 1904.)

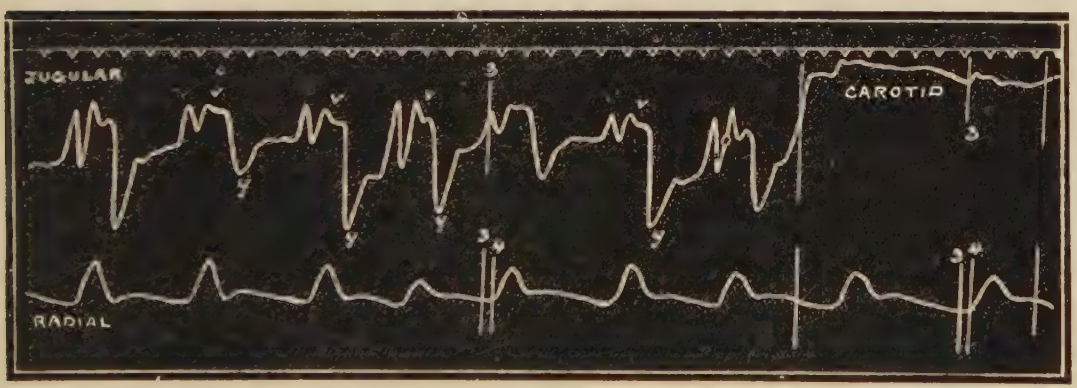


FIG. 110. The jugular pulse is of the ventricular form. (Observation 10, December 1906.)

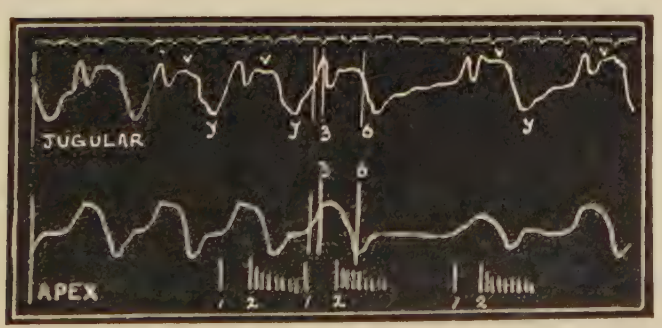


FIG. 111. The jugular pulse shows the same features as in Fig. 110. The shading shows only the presence of a diastolic murmur. Compare with Fig. 108. (Observation 10, February 1907.)

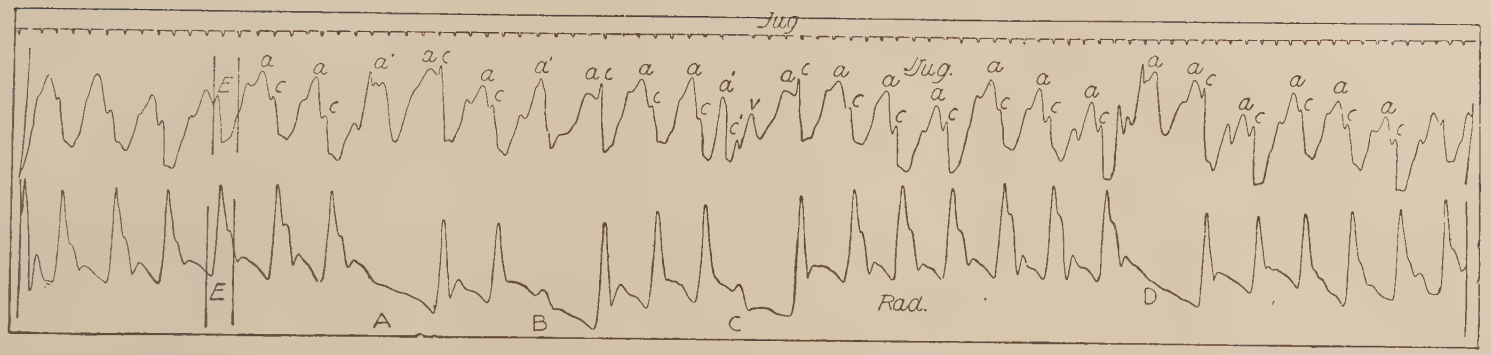


Fig. 112. Simultaneous tracing of the jugular and radial pulses, showing extra-systoles from three sources. The extra-systoles at *A* and *D* are probably from the same source as shown by the jugular waves. The extra-systoles at *B* and *C* are different in origin, that at *C* being an auricular extra-systole. Before the onset of auricular fibrillation (see Fig. 113). (Observation 11.)

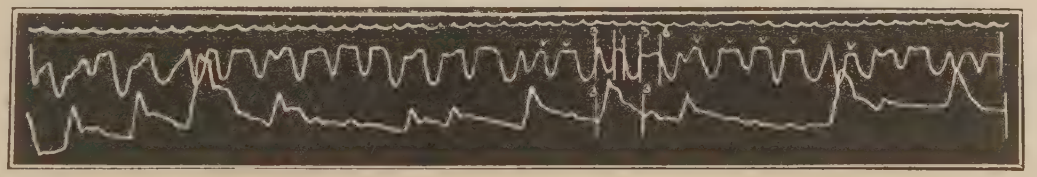


Fig. 113. Characteristic irregularity on the sudden onset of auricular fibrillation; the jugular pulse is of the ventricular form, and many of the radial beats are small and ineffective. (Observation 11.)

orthopnœa, and she died in 1907, aged 43. In the post-mortem examination there was found great stenosis of the mitral valves, while the apical half or two-thirds of the left ventricle showed extensive fibrosis. In the fibrotic tissue were nodules characteristic of the rheumatic condition.

OBSERVATION 11.—A female who had been under my observation for many years, whose pulse always showed the presence of extra-systoles of different kinds (Fig. 112), at the age of 54, in 1900 began to have short attacks of palpitation which tracings of the radial and jugular pulses showed were due to auricular fibrillation (Fig. 113). During the next three years these attacks continued to recur, sometimes lasting for several days. During the later attacks she

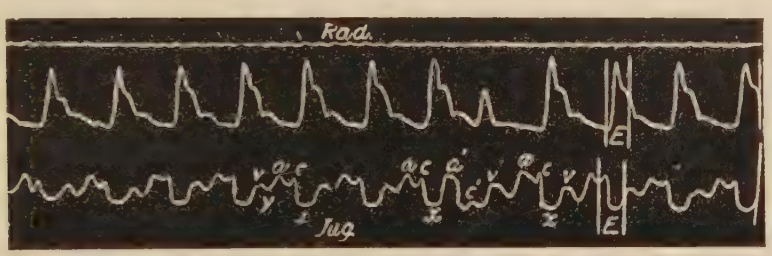


FIG. 114. Tracings after the cessation of auricular fibrillation (Fig. 113) show an extra-systole of auricular origin at a'. (Observation 11.)

at once became very distressed, with great weakness and shortness of breath, so that she was compelled to take to bed. A few hours after the onset of an attack the face became dusky, the lips cyanosed, the heart dilated, extending two inches beyond the left nipple line. There gradually set in great tenderness of the skin and deeper tissues over the chest. She could not lie down, but had to be propped up. There was also great tenderness over the liver, and the liver dullness extended two inches below the ribs. Œdema appeared over the ankles. The pulse was rapid and irregular, sometimes uncountable, and tracings showed the presence of imperfect beats (Fig. 113, also Fig. 61). When the heart reverted to the normal rhythm the patient's condition

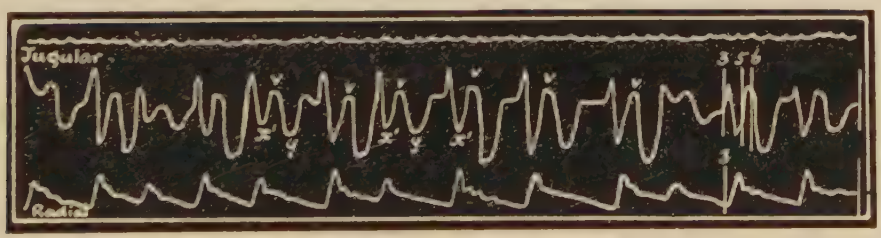


FIG. 115. After the auricular fibrillation became permanently established, the rhythm is irregular and the jugular pulse is of the ventricular type. (Observation 11.)

underwent a remarkable change. She was conscious of the heart becoming normal, and felt a great relief in the breathing, while within half an hour all the abnormal signs in the face and the enlargement of the liver disappeared, and in a few hours the heart was beating inside the nipple line. Within an hour of the cessation of the attack she would get up and resume her household duties. Fig. 114 shows a tracing shortly after the cessation of such an attack—regular rhythm in the venous pulse of the auricular type, showing one auricular extra-systole.

In October 1904 an attack began which never ceased till her death six months later. In addition to the former signs already described, there was towards the end extensive dropsy, ascites, and hydrothorax. Digitalis in this case had little effect, though it sometimes slowed the pulse a little (Fig. 115). Fig. 116 was taken shortly before her death.

At the post-mortem examination there was narrowing of the lumen of the coronary artery; no disease of the valves; the apical two-thirds of the left ventricle showed large patches of fibrosis.

OBSERVATION 12.—A female, 65 years of age, who had lived an active, vigorous life, was seized rather suddenly with shortness of breath. When I saw her she was propped up in bed, breathing rapidly and laboriously, the pulse small, rapid, and irregular. Next day she was quite well and out of bed, the pulse full and regular, 60 beats per minute (Fig. 117). The following day she again became very ill, the pulse small, rapid, and irregular, between 120-130 beats

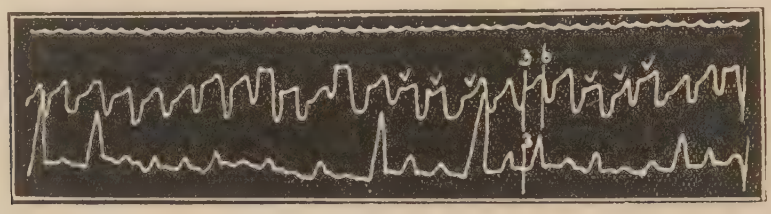


FIG. 116. Shows the character of the irregularity (auricular fibrillation) persistent one month before death. (Observation 11.)

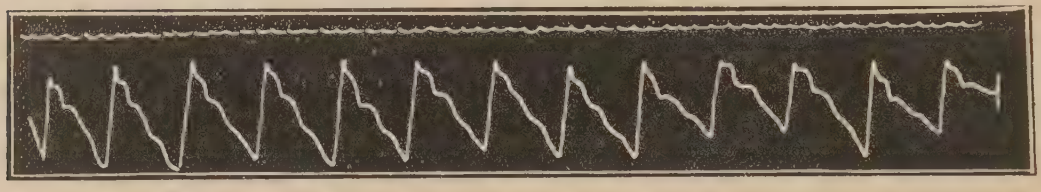


FIG. 117. Regular pulse between attacks of auricular fibrillation. (Observation 12.)

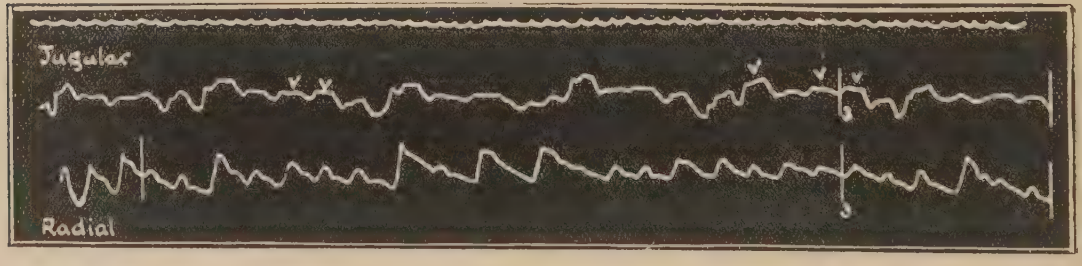


FIG. 118. Permanent inception of auricular fibrillation, leading to a fatal issue in three weeks. (Observation 12.)

per minute, with many imperfect beats (Fig. 118). In spite of a number of remedies which were tried, the heart dilated, the liver enlarged, extensive dropsy set in, and she died three weeks later.

OBSERVATION 13.—A man aged 58 had been suffering from slight pain over the chest, with great shortness of breath. Seven years before I saw him he was conscious of his heart beating irregularly, and had been more or less under treatment ever since by a great variety of drugs and other methods. During the last few months he had become very ill, and had to keep in bed, being propped up on account of the distressed breathing. Cheyne-Stokes respiration was present at times. The face was livid, the legs were swollen, and the liver enlarged. The radial pulse was 110 per minute, very irregular, with numerous small imperfect beats. The

heart's dullness extended one inch beyond the nipple line. The doctor in attendance said that digitalis had been often tried and was ineffective, but I found that only small doses had been given. The patient was given 30 drops of the tincture thrice daily, and in five days' time all the distressing symptoms disappeared, the pulse-rate had fallen. I saw him the following year, and found him wonderfully well—the heart was irregular, and the dullness extended to the nipple line. He was rather breathless, and had neglected to take the digitalis. A year later I found him much better, able to get about quietly by taking 5 to 10 drops per day of the tincture. He lived in comfort, though his powers of locomotion were limited, till he died at the age of 68.

OBSERVATION 14.—A male aged 64 suffered from attacks of pain in both arms, shoulders, and back when walking, especially after a meal. The pain began three months before I saw him, slight at first, latterly every time he walked over half a mile. The pulse was regular, 65 beats per minute. Seen again seven months later he complained no longer of pain, but of breathlessness, and he could walk only 100 yards. He had to be propped up in bed. His face was puffy and his ears were blue. The pulse was small and irregular, over 100 beats per minute. There was tenderness over the liver, and the liver dullness extended two inches below the ribs. Seen a month later there was no breathlessness, and he could lie flat in bed, but he was pulled up by pain in his arms when walking a short distance. The pain sometimes came on when at rest. The face was no longer puffy, and the cyanosis of the ears was gone. There was no tenderness over the liver, and the liver was normal in size. The pulse was 80 per minute and quite regular. He died suddenly a month later. At the post-mortem examination there was well-marked atheroma and calcification of the left coronary artery, with diminution of its lumen, and there were fibrotic changes in a considerable portion of the wall of the heart.

Paroxysmal attacks of auricular fibrillation.—A great many elderly people have frequent attacks lasting from a few minutes to a few hours. In some this causes no inconvenience, in others we meet with varying degrees of distress.

Before patients settle down to a permanent condition of auricular fibrillation there are periods when attacks of varying duration and of variable frequency may appear. One patient (aged 56), whom I had in hospital for three weeks, was wont to have attacks of auricular fibrillation lasting from half an hour to an hour and a half, four or five times in the twenty-four hours. The rate of the pulse was only slightly increased, and he was not conscious of any distress at any time.

The ill-health of the patient in Observation 11 began with short attacks of what she called palpitation, which were found to be due to short periods of auricular fibrillation, and were present at intervals for four years before the condition became permanent.

The patient in Observation 7 had the condition for a short time, and then, after six months' freedom, auricular fibrillation became permanently established, with no impairment of the heart's capacity.

OBSERVATION 15.—A healthy, active doctor, at the age of 47, after running 300 or 400 yards, had a heart attack which lasted two hours. The pulse was very irregular, about 90 per minute. He was conscious of a slight fluttering sensation, but suffered no distress of any sort. During the next four or five years he had several attacks when walking up a hill and after a full meal. These lasted from ten minutes to half an hour, but he was not conscious of any distress or limitation in his powers of walking. He led an active life till he was about 68, when his powers became gradually restricted on account of pain in his chest on exertion, and at the age of 71

his powers of walking are considerably limited on account of the pain in the chest, and the heart is quite regular.

OBSERVATION 16.—An active business man consulted me at the age of 44 for attacks of his heart 'thumping'. I saw him in one attack which lasted several hours, and the tracing showed the characteristic irregularity of auricular fibrillation. After a few weeks the attacks ceased, and he led an active, vigorous life until he was 59, when he had a severe attack of angina pectoris. After this he led a life at a somewhat lower level, experiencing pain across the chest when walking up hills. This pain ceased when he was 68 years of age, when he became breathless on exertion. I found the heart with the irregularity characteristic of auricular fibrillation. I saw him two years later when he was supposed to be suffering from a pneumonia because of great breathlessness, spitting of blood, dullness of the lungs at the back. The condition, however, was manifestly due to heart failure, and he promptly responded to full doses of digitalis. Two years later he had another attack of extreme heart failure, and he gradually sank and died at the age of 72.

OBSERVATION 17.—I was summoned in the middle of the night to see an old lady of 80 who was seriously ill. She was gasping for breath, and could not lie down; her face was livid, the lips blue, and the pulse scarcely perceptible, rapid, and very irregular. She was barely conscious. She was evidently moribund, and I told the friends to let me know in the morning when she died. As I did not hear from them, I called at the house in the course of the forenoon, and was told my patient had gone out for a walk. When she came in I found the heart quite regular and at a moderate rate.

CHAPTER XXX

The Origin and Nature of Auricular Fibrillation

Distinctive features of auricular fibrillation.
The disorderly activity of organs.
Disease of the s.-a. node.
The age incidence of auricular fibrillation.
The relation of heart-block to auricular fibrillation.
A comment on the origin of auricular fibrillation.

Before we can understand heart failure and deal with it intelligently it is necessary to know how auricular fibrillation is induced and the nature of the changes which follow its production.

Distinctive features of auricular fibrillation.—The main points which are to be found in the records of patients given in the last chapter are as follows:

1st. The sudden onset of a condition in which all signs of the normal auricular activity disappear, shown by the disappearance of the auricular wave from the jugular pulse and the change of the jugular pulse from the auricular type to the ventricular type, and by the disappearance of the presystolic murmur with persistence of the diastolic murmur.

2nd. The appearance of an irregularity of the heart peculiar and distinctive.

3rd. The exaggerated effects on the pulse-rate in response to effort, to rest, and to digitalis.

4th. The occurrence of heart failure of a particular kind in some, and its absence in others.

5th. The recovery from heart failure (*a*) suddenly on the return of the normal rhythm; (*b*) gradually but speedily under the influence of digitalis.

6th. The danger to life when there is myocardial degeneration.

The disorderly activity of organs.—In the classification of symptoms on p. 75, symptoms were shown to be due to an increased or decreased activity of the organ which exhibits them, or to a disorderly activity of the organ. The disorderly activity of an organ is illustrated by auricular fibrillation.

It has been shown that the functional activity of every organ is regulated and controlled so that its functions are performed in an orderly manner at once purposive and effective. This function of control is exercised by structures specially evolved for the purpose. If they fail to function, the organ may show functional activity, but it becomes ill-regulated or disorderly. It is manifest that to search for the source of this loss of control in the organ

which shows this disorderly activity would fail to reveal the source. The source must be sought for in the structures which are concerned in the control.

This principle can be applied to the condition called auricular fibrillation. The normal auricle beats in an orderly, purposive, and effective manner. In fibrillation the muscle fibres contract in a disorderly, purposeless, and ineffective manner. The conclusion to be drawn is that a disturbance has taken place which has removed the muscle cells of the organ from its controlling structures.

As has already been remarked, the auricle is not the only structure over which control is lost—the a.-v. node suffers the same loss and behaves in its own particular way.

The reasons for attributing the origin of such disorderly movements as occur in auricular fibrillation, to the loss of the influence of the s.-a. node, have already been discussed (pp. 106 and 161), and it is only necessary to point out some additional arguments in its favour.

Disease of the s.-a. node.—In using the term s.-a. node there is included the structures that connect this node with the muscle fibres of the auricle and with the a.-v. node. What these are is a matter of speculation. Suggestive evidence in favour of the cessation of activity of the s.-a. node is found in the consideration of the relation of organs to disease. There is no organ of the body exempt from disease, and there is no reason for assuming that this node is exempt.

The more active organs are particularly liable to disease. When we consider that it is the sino-auricular node which is the first part of the heart to be subjected to increased activity when effort is made and that it stands in a regulating capacity to the other parts of the heart, and also participates in the call upon the heart for more work, we will realize that it is exposed to circumstances that will test its integrity. Such an active organ must be richly supplied with blood, as all organs are dependent in their activity upon their blood supply, and we know that no organ is immune from arterial degeneration, or rather capillary obliteration, and a defective blood supply. We know also that the time when an organ shows its impaired function is when it is called upon to make an effort, because then it fails to respond with that ease and efficiency which it did when its blood supply was good and its efficiency unimpaired.

Accompanying an impaired blood supply, and probably due to it, we know that the cells of an organ diminish in number. According to the laws of fluctuation, such an organ's ability to respond to effort is distinctly limited, because when quiescent, fewer cells are resting, and so exhaustion in response to effort is more easily produced.

Bearing all these things in mind, we would expect that a proportion of elderly people would have degenerative changes in the blood-vessels and cells of the s.-a. node, just as they occur in other organs and other parts of the heart, like the a.-v. node and bundle. Moreover, the impaired function would more likely be shown at a time when the organ was stimulated to

increased effort, and it is in consequence of increased effort that fibrillation frequently starts. Indeed, the heart failure which sets in after 'overstrain' is almost invariably due to the onset of auricular fibrillation, while statistics show the great preponderance of auricular fibrillation among people up in years.

Assuming the s.-a. node to be damaged, how would the evidence of the damage be shown? The application of the principle of the reflex arc shows that the evidence will be revealed by the structures which are dependent upon the s.-a. node. Of these structures the auricle is the most closely associated, and any defect in the node is bound to be 'manifested' in the auricle. Moreover, there is no reason for assuming that fibrillation is an evidence of disease of the auricle. The ceaseless activity of the muscle cells in fibrillation should be far more exhausting than the normal activity, with its short periods of activity and its relatively long periods of rest. Patients may live for over twenty years and the auricle never cease fibrillating. It is a curious fact that nothing whatever is known clinically of affections of the auricle apart from the disturbances of the genetic system.

Another concept which helps to reveal the functional peculiarities of an organ is that the part played by an organ cannot fully be understood by studying it when it is acting normally, its peculiar functions being revealed only when its activity has ceased.

While we know that the s.-a. node regulates the rate of the heart, we cannot tell if it has any other effect unless we can see what happens when it ceases to function. In some respects it is like a nerve in that it controls the movements of muscles. When a motor nerve ceases to function—the muscle is said to be paralysed. But the muscle does not lie an inert body—as we have seen, it evinces a new kind of activity; it passes into fibrillation.

Moreover, there is the influence of control to which attention has already been called. It is a function difficult to describe or to recognize till it is lost. It is to this loss of control that some of the remarkable features in auricular fibrillation can be attributed.

The age incidence of auricular fibrillation.—In my practice as a general practitioner I was struck with the frequency with which I found auricular fibrillation in elderly people, and in younger folks who gave a history of rheumatic fever. To make sure of this relationship I have analysed 500 of my cases from among those that I had seen when in general practice and in consulting practice in London, unselected—taken as they appeared. These are tabulated in Fig. 119, which gives a fair representation of the age at which the condition is recognized by the doctor. The age which is taken here is that at which they first consulted me. It is therefore inexact, inasmuch as the condition may have been present for some time—weeks, and even years before I saw them. I tried to get from the patients some idea of the onset, but found it too unsatisfactory for use. The age taken serves the purpose of showing roughly the age incidence. I tried to separate those with a rheumatic history, and although the majority of those under 40 gave a definite

history of rheumatic fever, after that the evidences were too indefinite to be of use. A few of the younger cases gave no history of rheumatic fever.

An instructive diagram might have been made of the ages at which the patients died, and another showing the duration of the condition after the manner in which I analysed the cases of angina pectoris. But such diagrams would almost certainly lead to confusion, for the reason that people do not die of auricular fibrillation—it is only a condition that embarrasses the heart

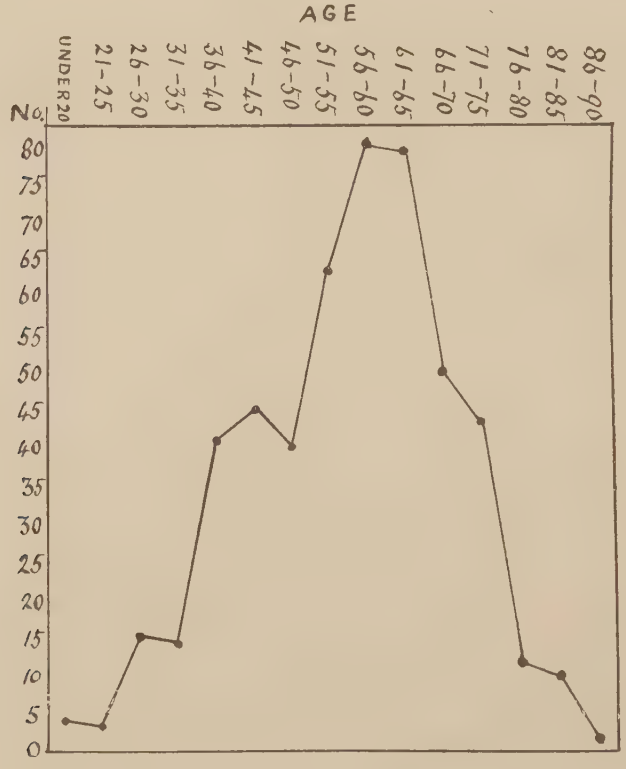


Fig. 119. Chart showing the age incidence of 500 cases of auricular fibrillation. The age stated is approximately that at which fibrillation started.

in its work. The failure of the heart which results in death is really due to changes in the heart muscle, which renders it incapable of carrying on its work hampered by this embarrassment.

The character of the curve has a curious resemblance to that of the deaths from angina pectoris after the age of 40. In the angina pectoris curve the steep rise to the maximum number occurs between 61 and 65, then there is a rapid fall in the numbers. This is practically the curve of the death-rate in general, and the reason for the fall in the curve in auricular fibrillation after 65 is simply due to the fact that there are fewer people living then.

The most common conditions associated with auricular fibrillation are

rheumatic fever and senile changes. It is worthy of note that these are also the conditions which are associated most commonly with partial and complete heart-block, and the suggestion arises that while the latter is undoubtedly due to an invasion of the genetic system—the a.-v. node and bundle—the former is likely to be due to the invasion of the s.-a. node. This aspect of the subject is discussed in Chapter XL.

A comment on the origin of auricular fibrillation.—The view held at present with regard to auricular fibrillation is that originally suggested by MacWilliam, which is that fibrillation is essentially due to some agent or condition which upsets the normal relation between the non-responsive or refractory period of muscle cells and the rate of conduction—that is, that the fibrillation of the auricle is supposed to be due to a shortening of the refractory period, and to be different from fibrillation of the voluntary muscles. Although this view has been accepted by various investigators, I have failed to find any evidence put forward in favour of its support beyond the assertion that the auricle simply passes into fibrillation. This absence of proof is a point to which I would like to draw attention. I have shown that fibrillation is but one of a number of events—the a.-v. node becomes altered in a remarkable manner, shown by its peculiar excitability, and there is produced the two peculiar forms of ventricular irregularity. No reasonable explanation for these changes is forthcoming from the supporters of MacWilliam's views. The statement that the fibrillation of voluntary muscle is different from that of cardiac muscle is, after all, nothing but an assertion. No one has taken the trouble to define the difference. On the other hand, I have searched for evidence, and find that all the movements of the fibrillating auricle are to be found in the fibrillating voluntary muscle. Even if there is a 'circus rhythm', that does not prove anything, indeed the circumstances in which this circus movement was first recognized suggest it to be a secondary condition, and to involve structures that have, so far, not been recognized in the auricle. It was found first in a ring of the body wall of the jelly-fish, and its production was only possible when this ring was removed from the control of the marginal sensory bodies. It was shown further that the circus movement immediately stopped when an impulse came from the sensory bodies. Fibrillation was shown also only to occur when the tissues were removed from the control of the sensory bodies (see p. 63).

I would like the reader to compare these and the other facts described in this book, which are produced in favour of the view I seek to establish that auricular fibrillation and all the other phenomena result from the loss of the controlling influence of the s.-a. node, with the bald assertion that fibrillation of the auricle is an independent condition.

A further reason I have for presenting the matter in this way is that I am strongly of opinion that we must, if progress is to be made, get a knowledge of the laws or principles that govern the production of all clinical phenomena. One of these which has been enunciated is that the activity of an organ is always dependent upon certain structures that regulate and control it. Any

modification of this activity is not due to the structure that exhibits the activity, but to some disturbance in the processes which regulate and control its activity. This was shown to apply to fibrillation in voluntary muscle, to fibrillation and the circus movement in the jelly-fish, and it affords an explanation for every other form of cardiac activity. It requires, therefore, some very potent argument to show that auricular fibrillation is either an exception to this law, or that the law is not tenable.

If the law is held to be untenable it is then the duty of the advocates of the contrary view to demonstrate some other law by which fibrillation can be shown to have an affinity to other natural phenomena. Otherwise, we fall back into the old illogical method of looking for a different cause for each and every phenomenon.

CHAPTER XXXI

The uncontrolled Auriculo-Ventricular Node

The independent a.-v. node.
The effects of loss of control.
The relation of the a.-v. node to the fibrillating auricle.
The a.-v. node in the tortoise heart.
Stimulation and inhibition of the a.-v. node.
The value of the sphygmogram.
The pulse irregularities of auricular fibrillation.
The a.-v. node and the contraction of the ventricle.
The nature and origin of the small beats.
The disappearance of the small beats.
The relation of heart failure to the small beats.
The imperfect control of the independent a.-v. node.

The independent a.-v. node.—In the normally acting heart all the elements of the genetic system act so harmoniously that it is not possible to recognize the peculiar properties of each element. The influence of the sino-auricular node is so subtle and far-reaching that its effect cannot be recognized until its influence is lost. The a.-v. node then takes on the function of the s.-a. node, and an opportunity is afforded of studying its peculiarities. When the influence of the s.-a. node is removed the a.-v. node becomes altered in a remarkable manner, and affords the opportunity for the observation of living processes to be found in no other structure in the body. It may be said that through it we get a glimpse into nature's workshop, and can recognize factors at work which reveal the processes concerned in the activity of every organ. Its study shows the manner in which ventricular contractions are regulated and controlled so as to meet the calls made upon the heart in the circumstances of daily life, and it also shows how the ventricular contraction can be modified to produce heart failure.

The effects of loss of control.—In the comparison between the effects produced by section of a nerve to a voluntary muscle and the cessation of the function of the s.-a. node, there are some resemblances and some differences, and the effect of the loss of influence which occurs on the section of a nerve only extends to the muscle supplied by the nerve. The influence of the s.-a. node extends beyond the auricle, to the a.-v. node, so that when the s.-a. node ceases to exert its influence three results follow which affect the a.-v. node: (1) It becomes more susceptible to stimulation by the sympathetic

and vagus nerves; (2) it is subject to a shower of impulses from the auricle; and (3) it does not meet the requirements of the body like the s.-a. node.

The relation of the a.-v. node to the fibrillating auricle.—As the ventricular contraction is dependent upon the a.-v. node we can gather what is happening in the node by the behaviour of the ventricle.

In the fibrillation of the voluntary muscles no result can be detected beyond the slight movements that may be present in the muscle, for the reason there is no other structure on which the impulses produced by the fibrillating muscle can act so as to produce a perceptible effect. In the heart, on the other hand, the a.-v. node lies in the auricular wall, and it is exposed to the impulses which the fibrillating auricle throws out. The a.-v. node responds to these impulses in a variable manner. If it is in an excitable state, it responds at a frequent rate, as shown by the rapid pulse. If it is in a much less excitable state it responds infrequently, as shown by an abnormally slow pulse. As a consequence, we find that agents that increase the excitability of the a.-v. node cause a greater increase in the pulse-rate than normal, while agents that decrease the excitability cause a greater decrease in the pulse-rate.

The a.-v. node in the tortoise heart.—The a.-v. node differs so remarkably in its activity when it escapes from the control of the s.-a. node that it is of interest to investigate its behaviour under all circumstances. Gaskell describes some peculiar effects in his investigations into the tortoise heart.

In the canalis auricularis of the tortoise there is a ring of muscular fibres of a peculiarly sensitive kind. Gaskell[1] describes the manner of exploring this ring as follows:

'Slit up one auricle and the ventricle, and pin back so as to expose the septum; the two vagi nerves on the septum, with their terminations in Bidder's ganglia, are then most easily seen, as well as the muscular ring between auricles and ventricle, and the whole field can be explored, under a lens, with the point of a needle before and after removal of the septum and Bidder's ganglia. Now, CUT AWAY THE SINUS, the whole preparation remains quiescent; touch the outlying auricular or ventricular tissue with the needle point, each time a single contraction takes place, and no more; touch either of Bidder's ganglia, not a single contraction takes place; pierce it again and again, and pull it about, no sign of contraction. *Touch the auriculo-ventricular ring of muscle with the slightest stimulus, immediately a series of rhythmical contractions occur. It is most striking to see, after removal of the septum, how every portion of auricular and ventricular tissue can be explored up to the very edge of the ring, without obtaining more than a single contraction, while immediately the needle touches the muscular ring a series of rapid contractions results.* Conversely, the two Bidder's ganglia can be cut away, and yet stimulation of the ring will produce a rhythmical result.' The italics are Gaskell's.

I have assumed that this muscular ring is the progenitor of the auriculo-

[1] Schafer's *Text-book of Physiology*, vol. ii. p. 179.

ventricular node, and here, when the control of the sinus is gone, it behaves in some respects like the auriculo-ventricular node in auricular fibrillation.

Stimulation and inhibition of the a.-v. node.—Before entering upon the descriptions of the variable activity of the a.-v. node it is necessary to refer again to some facts concerning stimulation and inhibition. In discussing these variations in activity, they were shown to depend upon two factors—the excitability of the cells and the number of impulses. In the a.-v. node in auricular fibrillation we find an excellent opportunity of investigating the relation between these two factors.

The muscle cells of the fibrillating auricle are in continuous activity, and impulses are being continuously produced, and as a consequence there is thrown upon the a.-v. node a continuous shower of impulses. The variations in the activity of the a.-v. node can thus be referred, not to a variation in the number of impulses, but to variations in the excitability of its cells. A doubt may be thrown upon the number in the continuous stream of impulses from the fibrillating auricle, but, as we shall see presently, in auricular flutter we can demonstrate that the numbers are constant.

It must be borne in mind that the activity of the a.-v. node can be recognized by the number of pulse-beats, the contraction of the ventricle being as dependent upon the node as the voluntary muscle is upon its nerve. The pulse-rate then is taken as a measure of the activity of the a.-v. node, and the circumstances that increase or decrease its activity, or in other words its stimulation and inhibition, can be studied.

In dealing with the application of the principle of the reflex arc, the curious susceptibility of the a.-v. node was demonstrated and commented on (see Figs. 7-9, pp. 45 and 46). Similar effects are found in auricular fibrillation.

In Observation 9 it was shown that bodily effort caused a great increase in the pulse-rate. This is brought about by impulses arising in the muscles of the body which are conveyed to the central nervous system and thus stimulate the sympathetic or diminish the effect of the vagus (p. 62). This effect results in an increase of the excitability of the cells of the organ to which the nerves are distributed. The organ in this case is the a.-v. node, and the result of such stimulation is seen in the increased rate of the ventricle. When effort ceases and impulses no longer arise in the muscles, the effect of the increased excitability of the cells of the a.-v. node does not pass off at once, but gradually, and this gradual decrease in the excitability of the cell is to be seen in the decrease of the rate of the pulse.

From the consideration of the records shown in Figs. 106 and 107 we observe that while the impulses are constant in number the activity of the a.-v. node varies remarkably, and this variation can only be due to the effect of the vagus and sympathetic nerves upon it.

In discussing the matter of reciprocal innervation, it was shown that an increase or decrease in the rate of the heart while both sympathetic and vagus nerves were intact, could not be attributed entirely to one or the other, but that probably when an agent stimulated one, it at the same time decreased

the activity of the other, so that an increase of rate may be due to the diminution of vagal activity, as well as to an increase of sympathetic activity. The slowing, on the other hand, may be due to a diminution of sympathetic activity and an increase in vagal activity.

It will be observed in these instances that the increased rate was much greater than occurs in the normal heart with the same amount of effort, while the fall of the rate so soon after rest was much greater than occurs in the normal heart. This difference of reaction is one of the reasons which leads me to suggest that the s.-a. node exercises an influence upon the a.-v. node.

The suggestion arises that the s.-a. node regulates and controls the ventricular contraction through the a.-v. node. When this influence is lost the discharge of impulses from the a.-v. node which stimulate the ventricle is irregular.

This leads to the consideration of the peculiar pulse-beat which appears in auricular fibrillation.

The value of the sphygmogram.—Of late years there has been a tendency to neglect the sphygmogram in the investigation of the heart. In polygraph tracings when tambours and air conduction are used, the radial pulse is no doubt often taken as a record of the rate and rhythm of ventricular contractions, but little attention has been paid to the variations in the size of the beats and their significance. It is true that careful records are sometimes obtained in this way, but the method requires a good deal of experience to enable one to get a satisfactory record, while in many pulses, and particularly those where it is of importance to get a good record, the method is not adequate for the purpose.

In addition to various methods of the conduction of the pulse-beat by air from tambour to tambour, I have used the direct methods of Marey, of Dudgeon, and of Von Jacquet, the latter being a modification of the Dudgeon. Of all these the Dudgeon is by far the most serviceable.

The value of the sphygmogram lies in the fact that a good record is an accurate representation of the output of the left ventricle. It is not possible to measure the value of the waves, but still valuable information is obtained in that it shows accurately the variations of the output in irregular action of the ventricle.

The sphygmogram, therefore, gives information of a kind that nothing else does except the finger of the doctor. By comparing the impressions made on the finger and the character of the sphygmogram, information of a useful kind is obtained, and in the course of time the doctor becomes so educated that he can trust to his finger to detect some of the evidences of the cardiac condition that can be obtained in no other way.

The pulse irregularities of auricular fibrillation.—When a great number of pulse tracings are analysed, each pulse-beat of the normal regular heart presents identical features, while in those that are irregular in rhythm, the individual pulse-beats present different features. Most irregularities show pulse-beats which vary in size according to the length of the pause that preceded them,

so that it can be laid down as a rule that the size of the beat varies according to the length of the preceding pause. The reason for this is twofold: first, the longer pause permits a greater amount of blood to accumulate in the ventricle; and second, the longer pause permits of a greater number of muscle fibres participating in the contraction. This relation of the pulse-beat to the preceding pause is found in irregularities which occur in variations of the activity of the s.-a. node, as in the youthful type of irregularity, Figs. 21-23, also in partial heart-block, and in one of the forms of auricular fibrillation, Fig. 91.

There is in auricular fibrillation another form of irregularity of a disorderly kind in which the pulse-beats vary in size without any relation to the length of the pauses which precede them, large beats follow a short pause, and small beats follow a longer pause, as shown in Fig. 120. This kind of pulse occurs in no other condition than auricular fibrillation.

In auricular fibrillation the irregularity of both forms is also characteristic in that the length of the diastolic period is continually varying, whether the

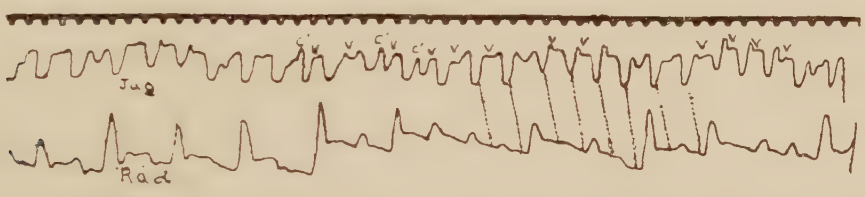


FIG. 120. Tracing showing the irregularity characteristic of auricular fibrillation. There are numerous small imperfect beats, and there is no relation in their size to the length of the preceding pause.

rate is slow or rapid. Occasionally, when very slow the rhythm is fairly regular.

In auricular fibrillation we have therefore got two forms of irregularity, one in which the size of the beat is related to the length of the preceding pause (Fig. 91), and another where this relation does not hold true (Figs. 120 and 121). The latter condition occurs chiefly when the rate of the heart is considerably increased. When the patient is at rest, the pulse may be slow, and the irregularity shows that the size of the beat has a relation to the length of the preceding pause.

The great increase in rate which results on effort is due chiefly to the increase in the number of those imperfect beats whose size has no relation to the duration of the preceding pause (Figs. 106 and 107). This division of the pulse into two forms is of value in recognizing the nature of heart failure and the action of drugs and exercises. It is necessary to understand the nature of the small beats in the pulse, and to do this we must first consider in more detail the relation of the a.-v. node to the contraction of the ventricle.

The a.-v. node and the contraction of the ventricle.—In the description given of the s.-a. node it was shown that the ventricle varied the force of its beat according to the demands made upon it. Such a variation is common

to all muscular organs. Organs like the bladder, or the bowel, contract with varying force. If an adequate stimulus arises, such as a stone in the bladder, or obstruction of the bowel, the force of contraction becomes greatly increased. The hand also will close with slight force or great force. All these results come through the number of the muscle cells engaged in the contraction.

There are good grounds for assuming that all muscular organs are thus graded in their action, so that at one time more muscle cells are engaged in contraction than at other times. It may not be possible to make out this graded contraction in such an organ as the heart, but seeing that its work necessitates a variation in its activity, the presumption is that it acts like other muscular organs.

The structures that regulate and control the contraction vary in different organs, depending partly or entirely upon the manner in which the organ was evolved. In the brief description of the development of the heart it was shown that the genetic system was evolved in a peculiar manner, different, in all likelihood, from the evolution of any other regulating structure.

Whatever be the nature of these regulating structures, they all control the muscle in the same way, and bring about the same kind of effect, and it is therefore reasonable to suppose that as the motor nerve regulates and controls the force of contraction of a voluntary muscle, the a.-v. node regulates and controls the force of the contraction of the ventricle. Under normal circumstances it is difficult or impossible to make out the peculiar function of the different parts of the regulating system of the muscle of the heart. But when a disturbance arises, the peculiar part played by one or more of these structures comes to light, as has already been pointed out in the production of auricular fibrillation. The peculiar pulse-beats in auricular fibrillation shed a light upon the function of the a.-v. node. We have seen that the beats vary in size, and that the size does not depend upon the length of the preceding pause, so that the conclusion is arrived at that the size of the beat depends upon the number of fibres of the ventricle taking part in the contraction. As the ventricle responds only to impulses from the a.-v. node we are therefore forced to the conclusion that the size of the beats is dependent upon the number of impulses which the a.-v. node discharges at a given time.

According to this view there is a difference in the time of recovery of the cells of the a.-v. node, so that when a portion of the node discharges impulses a portion of the ventricle contracts. The portions stimulated seem continually to vary, and this tendency for separate portions to act at different times becomes greater with the increased excitability induced by bodily effort.

When the increased excitability ceases with slowing of the rate the cells act more together.

The nature and origin of the small beats.—This explanation implies the contraction of a variable number of fibres at different times. Though the electro-cardiogram is the expression of ventricular contraction it throws no light upon the number of fibres taking part in the contraction.

We can, however, reasonably infer a good deal from it when taken in

relation with the output as shown by the radial pulse. It must be remembered that though the electro-cardiogram throws no light upon the size or character of a pulse-beat, it does give information of the manner in which the wave of contraction spreads through the ventricular muscle. Normally the impulses enter by the a.-v. bundle, and the contraction spreads in a particular way. The impulses in auricular fibrillation enter by the same path, and the contraction spreads in the normal way. We must, therefore, infer that the

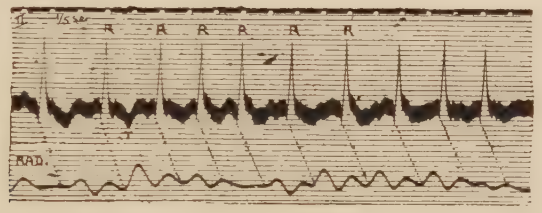


Fig. 121. An electro-cardiogram of the heart from a case of auricular fibrillation taken at the same time as a tracing of the radial pulse. The complex of the electro-cardiogram is indicative of that given by the ventricle when the stimulus for contraction comes from the auriculo-ventricular node. The radial tracing is typical of the small imperfect beats which occur in auricular fibrillation, and resemble those in Fig. 120. (J. Parkinson.)

contraction of the ventricle is not due at one time to a small portion contracting here, at another time a small portion contracting there. The wave of contraction follows the usual path through the ventricle, affecting a varying number of fibres as it passes in the normal sequence. In an ordinary contraction the parts follow one another in a particular sequence, whether it is a weak contraction of a few fibres or a strong contraction of many fibres. This seems to be the method common to all muscular organs as shown in the peristalsis of the bowel, and in the voluntary muscles, as when the hand closes

Fig. 121a. A diagram of the events in Fig. 121.

completely, but gently or firmly, *i.e.* when a few fibres or a great many fibres participate.

In auricular fibrillation all the beats, big and little, after short pauses or long pauses, produce the same type of electro-cardiogram (Figs. 121 and 121a). From this we infer that not only did the impulse arise in the a.-v. node and pass by the a.-v. bundle, but the order of contraction was the same as that which occurs in the normally acting heart.

This, which seems to be a correct interpretation, has a profound meaning. It shows that the impulses which pass to the ventricle are conveyed by separate strands going through the whole course of the ventricle, and causing it to contract in a strong or a gentle manner, according to the number of fibres,

just as the neurones which cause contraction of a muscle in a strong or a gentle manner along the whole course of the muscle.

This can be represented in a diagram (Fig. 122), where the nerve is represented by two neurones, or the bundle by two strands. Each neurone or strand breaks up into a number of branches, represented by two branches in the diagram, each of which supplies muscle fibres, so that each neurone supplies fibres along the whole length of the muscle. Impulses conveyed by one neurone contract the whole muscle, but gently. Impulses conveyed by two neurones or strands cause a stronger contraction, while impulses conveyed by more neurones or strands cause a still stronger contraction. But in each case the shortening is complete.

Fig. 122. Diagram showing the distribution of two neurones to a muscle, with fibrils of each neurone being distributed to every part so that a complete contraction can occur in response to the stimulus from a few neurones in a nerve. Stronger contractions result from the employment of a number of neurones.

The disappearance of the small beats.—If we compare the tracings of the radial pulse when a patient suffers grievously from heart failure, with the tracings when the heart's strength has been restored and the signs of heart failure have disappeared, it will be found that the small beats which were present during the heart failure entirely disappear when the heart's strength recovers. This recovery takes place both when the normal rhythm returns, as shown in Observation 11, or when the small beats disappear under the influence of digitalis, as shown in Observation 20.

The relation of heart failure to the small beats.—These observations suggest that the small beats are concerned in the heart failure. This view is strengthened by the fact that in those cases where auricular fibrillation sets in with an absence of the small beats there is little or no limitation in the heart's strength. This is well brought out in Observations 5 and 7, where it was shown that the onset of auricular fibrillation was not accompanied by any sign of heart failure.

The imperfect control of the independent a.-v. node.—On p. 62 reference was made to the differences between the s.-a. node and the independent a.-v. node and the independent bundle. It was shown that these two last-named structures had not that faculty of accommodating their activity to the requirements of the body, which was possessed by the s.-a. node through

its relation to the sympathetic and vagus nerve. This lack of accommodation is well brought out in Figs. 106 and 107, from Observation 9. There it was shown that a slight effort which in a normal acting heart would raise the pulse-rate 5 or 10 beats, induced a rate of 170, most if not all the beats being of the ineffective kind.

This is a most important point which requires to be grasped in order that the nature of the heart beat and the principles of treatment be understood. It shows how it is that effort induces heart failure, and to prevent heart failure steps have to be taken to protect the ventricle from the excitability of the a.-v. node.

CHAPTER XXXII

The Production of Heart Failure in Auricular Fibrillation

Heart failure in auricular fibrillation.
The mechanism of heart failure.
The symptoms of heart failure.
Sudden recovery from heart failure.
Angina pectoris and auricular fibrillation.

Heart failure in auricular fibrillation.—In a great many people the heart is capable of maintaining an efficient circulation when the body is at rest, but when exertion is made the heart cannot maintain an efficient circulation and signs of heart failure are produced, such as pain or breathlessness.

The reason for this is very simple. At rest few muscle fibres are sufficient to maintain an efficient circulation ; on effort a call is made for more fibres to participate in the contraction. In the normal heart sufficient fibres are in reserve and respond to the call. When the fibres are diminished in number as a result of disease, the heart cannot respond to the call, and the circulation becomes inefficient, so that signs of heart failure arise. This seems such a reasonable explanation that it does not need elaboration. But while it is true of the manner in which heart failure takes place in auricular fibrillation, the failure is brought about so peculiarly that it requires further consideration, particularly as we have the means of restoring the circulation in a way that does not often arise under other forms of heart failure.

In speaking of the manner in which the hand closed gently, it was reasoned that although the muscles were completely shortened only a few fibres participated in the active contraction. It was also reasoned that the normal contraction of the ventricle would take place when only a portion of the fibres participated, and even when these took place the chamber was completely emptied and the resultant pulse wave was full and of good strength.

The small pulse waves in auricular fibrillation are evidences of great weakness of the contraction of the ventricle. This can only be due to the participation of fewer muscle fibres in the contraction.

The mechanism of heart failure.—The small beats of the pulse with a great increase of rate do not throw out as much blood as the big beats, nor does the contraction of the ventricle exercise the same force on the blood in the arteries. If we assume for illustration that when the pulse-beats are about 60 per minute the systolic blood-pressure is 140, and the diastolic 80,

each contraction of the heart would be adding to the arterial pressure 60 mm. Hg. If the rate is exactly doubled, and the blood-pressure of the beats falls to, say, 100 mm. Hg. in the same period, the two beats represent a force exercised by the ventricle of 200 mm. Hg. in place of the 140 when there was but one beat. If the diastolic pressure remains at 80 then the force exerted upon the arterial blood is 40 mm. Hg. That is to say, that in the same period of time, when the pulse-rate is 60 per minute, a force of 140 mm. Hg. is spent by the ventricle, of which 60 is added to the force which moves the blood in the arteries. In the other case during the same period of time 200 mm. Hg. is expended and only 40 mm. Hg. is added to the force which moves the blood in the arteries.

This illustration is not put forward as being mathematically correct, but merely to enable us to understand the nature of heart failure.

The symptoms of heart failure.—With the onset of fibrillation different people show very different effects as shown in the cases recorded. In some cases heart failure speedily follows the onset of auricular fibrillation. Fig. 113 is a pulse tracing taken within a few hours after the onset of an attack. It will be seen how small and imperfect are the beats. In this patient the signs of heart failure described on p. 177 set in at once (Observation 11).

It is manifest that in this case the force which was required to send the blood round the circuit was insufficient, so that when the blood reached the venules it stagnated, as shown by the blueness of the lips and the lividity of the face. The portal blood to the liver goes through two capillary fields so that in the liver the force of the ventricle is felt less than in any other organ, and the stagnation of blood becomes greatest, and in consequence the liver greatly enlarges.

Sudden recovery from heart failure.—Striking as are the phenomena which are produced within a short period of the onset of auricular fibrillation, the results which follow its disappearance are equally striking (see p. 180).

The description given of heart failure and recovery is intended to bring out the essential features of heart failure in auricular fibrillation and the manner in which the heart's strength is restored. The recovery of the heart is seen to be due to the participation of a greater number of fibres in the systole of the ventricle—a result brought about in this case by the resumption of the normal rhythm.

In patients like those described in Observations 8 and 9, the rhythm does not become normal, but rest and digitalis produce great slowing of the rate, with disappearance of all the small beats. The great improvement of the circulation can be attributed to the increased strength of the individual beats, and this is brought about by an increase in the number of fibres taking part in the contraction.

From this point of view the heart failure in auricular fibrillation is brought about by the weak ineffective beats of the ventricle. It is necessary to inquire how these beats come to be so weak, as they are usually associated with a greatly increased rate. It is necessary also to inquire how they happen.

Angina pectoris and auricular fibrillation.—The conditions which are concerned in the production of these two phenomena and the symptoms which result afford an interesting and instructive study in symptomatology and in the manner in which heart failure is brought about. In the discussion of angina pectoris it will be shown that it arises through the muscle cells being called upon to do more work than they are fitted to accomplish, and that the regulator of the muscle takes no notice of the capacity of the muscle, just as the brain may force the muscles of the leg to do more work than they are capable of doing. The s.-a. node as the regulator may send impulses to the ventricle to accomplish work which it cannot perform. When this happens the overworked muscle gives rise to evidences of exhaustion, such as pain and the other phenomena characteristic of angina pectoris.

In patients suffering from angina pectoris auricular fibrillation may appear. When this happens pain is no longer experienced, for the muscle is not exhausted in the same way. One would imagine that the greatly increased rate in auricular fibrillation would hasten the exhaustion of the muscle, but the ventricular muscle is no longer under the control of the s.-a. node, but is under the control of the a.-v. node. We have seen that when under the a.-v. node the whole of the muscle is seldom called into activity, a few fibres acting at one time, and other fibres acting at other times. A consequence of this is that the feeble beats are due to the participation of but a few fibres at one time, and consequently extreme exhaustion of the heart muscle is never reached.

As has been shown the output of blood is so defective in auricular fibrillation that bodily effort is greatly restricted on account of the distress in breathing which results. It is no uncommon thing to find a man stating that he was pulled up on walking 400 or 500 yards because of the pain across his chest, but that now he is pulled up after 100 or 200 yards on account of distress in breathing. Such a statement almost to a certainty indicates that the patient now suffers from auricular fibrillation.

CHAPTER XXXIII

Prognosis of Auricular Fibrillation

The principles of prognosis in auricular fibrillation.
Variable results of auricular fibrillation.
The condition of the muscle.
Valve disease.
Response to treatment.
The degrees of heart failure.
Sudden death.

The principles of prognosis in auricular fibrillation.—The application of the principles for assessing the value of symptoms described in Chapter III. enables us to recognize the features which afford a prognosis in this condition. It was there shown that the basis of a prognosis is to be found in the capacity of the heart muscle to maintain an efficient circulation. Looking upon auricular fibrillation as being due to an affection of the s.-a. node, we recognize the prognosis can only be considered from the effects it produces on the functional capacity of the heart muscle. The fibrillation in itself is not a condition which imperils the life of the individual, for the functional efficiency of the heart depends on the ventricle, and not on the auricle, and as a matter of fact, many people lead vigorous lives who have had fibrillating auricles for many years.

When the s.-a. node ceases to exercise its influence, the a.-v. node is freed from its influence and takes up the function of starting the contraction of the ventricle.

This does not imperil life, but the a.-v. node, in stimulating the ventricle to a rapid rate with ineffective beats, hinders the ventricle from performing its work in an efficient manner.

But even when this happens, though the capacity of the heart may be limited, the life of the patient is not imperilled unless there is a disease of the muscle fibres of the ventricle. Then the embarrassment of the ventricle becomes so great that heart failure of a grave kind may follow. Thus the essential question in every case is the condition of the muscle of the ventricle.

Variable results of auricular fibrillation.—The observations cited in Chapter XXIX. showed great differences in the relation between auricular fibrillation and heart failure. In some the onset of auricular fibrillation caused little or no disturbance to the circulation, while in others life was imperilled from its onset.

It is necessary to appreciate why this is so, in order to obtain a prognosis, and to treat our patients in a rational manner. In no instance is it more necessary to bear in mind the dictum, which has been repeated again and again in this book, that disturbances of rhythm or rate are rarely in themselves of grave significance. The significance depends upon the functional capacity of the ventricular muscle.

The condition of the muscle.—In order to obtain a sound prognosis, the source of danger and the symptoms which indicate danger have to be recognized. In the description of the patients in Chapter XXIX. the symptoms of heart failure are clearly given, and in the patients with heart failure who did not respond to treatment there was found at the post-mortem examination extensive fibrotic degeneration of the muscle of the ventricle. The statement, therefore, can be made that with signs of advanced failure, resistant to treatment, in the absence of valvular disease, there is a damaged heart muscle.

Valve disease.—Other conditions tend to impair the efficiency of the ventricle by their presence. The chief of these are diseases of the mitral and aortic valves. The diseases of the mitral valve are the most common, as we find auricular fibrillation frequently occurring in people with mitral stenosis. Indeed, the termination of life in people with mitral stenosis is frequently preceded by auricular fibrillation, so that we can with fair assurance state that the conditions which produce mitral stenosis also favour the occurrence of auricular fibrillation. The condition which produced the valve damage also favours the occurrence of changes in the left ventricle, as in Observation 10 (p. 174). Auricular fibrillation, mitral stenosis, and damaged ventricles occur in different combinations. There may be mitral stenosis of extreme degree, terminated by death without fibrillation, or there may be mitral stenosis of a very slight degree with fibrillation with the ventricle gravely damaged, and there may be auricular fibrillation with great mitral stenosis with very slight damage to the muscle.

The part played by auricular fibrillation is that under certain circumstances it adds to the further embarrassment of the heart, but only when the rate is greatly increased with numerous imperfect beats.

Aortic regurgitation is also sometimes complicated with the onset of auricular fibrillation, and generally it is a grave combination, for it occurs chiefly in patients in whom the heart has already reached the stage in which there is a considerable limitation of its power to respond to effort, and the addition of this abnormal rhythm gravely embarrasses the heart muscle, while the imperfect beats are more ineffective on account of the regurgitation.

Response to treatment.—The fact that the heart failure can be kept in check in many cases enables us to use the response of the heart to treatment as a ground for prognosis. It has been shown that the rapid rate and imperfect beats are the immediate cause of heart failure, in patients with ventricles badly damaged and slightly damaged—if damaged at all. The degree of

recovery depends upon the extent of the damage to muscle, if the rate can be decreased, and the small beats made to disappear. It is always best to wait the result of treatment before giving a prognosis in patients with advanced heart failure—the more complete the recovery the more favourable the prognosis.

The degrees of heart failure.—The prognosis is favourable in those who have little or no limitation, and can go on without treatment. Many are able to lead useful lives for many years when there is considerable limitation of the response to effort, and who are not susceptible to digitalis. Others benefit so much by the continuous use of the drug that they can follow their occupations free from distress, and also carry on for many years. Others lead somewhat crippled lives for many years, liable to break down from heart failure, shown by breathlessness and dropsy, but restored by the use of digitalis. When digitalis in such cases is skilfully employed, the attacks of heart failure of an extreme degree cease, and the patients can lead useful lives at a lower level.

There are patients in whom the heart failure at first is fairly considerable, shown chiefly by shortness of breath on effort, sometimes with mental disquiet on account of consciousness of the heart's abnormal action. The rate may not be excessive and digitalis has not much effect, but, after a few months without any alteration in the heart symptoms, the patient becomes more composed and is able to make more effort, and by recognizing his limitations may lead a useful and contented life for many years in comfort.

Some patients become very rapidly ill in the course of a few days or a few weeks, with the heart rapid and irregular. Those who do not respond to treatment speedily sink and die within a few weeks or a few months of the onset of auricular fibrillation.

Sudden death.—In rare instances I have found the onset of auricular fibrillation accompanied by the loss of consciousness for a period of a few minutes. One can understand the reason for this by a glance at such a tracing of the radial pulse as shown in Fig. 116, where there is evidently a great diminution in the output of the heart. One such patient died within three months of the onset, irresponsive to all forms of treatment.

OBSERVATION 18.—This patient had an attack of loss of consciousness. I saw him two days later, and found the pulse rapid and irregular with many small beats. He was 65 years of age, and was a fairly active man for his years. One morning, shortly after rising from his bed, and while shaving, he felt faint, and making his way to his bed he fell down and became unconscious. When consciousness returned he found himself lying on the floor, and crawled into bed feeling very prostrate. He gradually regained some strength. I saw him two days later, as mentioned. Three days after I saw him his doctor reported to me that his heart-rate had fallen to 70 beats per minute, had become quite regular, was of good strength, and the patient felt remarkably well. Next morning he suddenly died. I take it that fibrillation had recurred, and the output of the heart was so small and the diminished output lasted so long that he never recovered consciousness.

The sudden death, however, may have been due to ventricular fibrillation, which is not an uncommon cause of death in such patients.

OBSERVATION 19.—In one patient with mitral and tricuspid stenosis, who had also suffered from angina pectoris, auricular fibrillation occurred, but the patient experienced no limitation when this occurred, the pulse-rate not having increased, being between 60 and 70 beats per minute. Four days later, when getting out of bed, she fell down dead.

In order to retain a proper perspective it should be recognized that these grave and fatal cases are very rare. Sometimes the onset is preceded by evidences of great cardiac weakness, such as angina pectoris.

I have already said that there are a great many individuals with fibrillation who lead useful and energetic lives, and whose capacity for work is little, if at all, impaired by the new rhythm. In such the prognosis is distinctly good.

The effects of auricular fibrillation are so varied that a clearer insight is gained by looking at each case from the point of view of the response to effort, not, however, ignoring the details, but giving them their due consideration. It is in estimating the reserve power that we get the most valuable information. It may be taken for granted that if distress is induced by exertion, so long as the exertion is persisted in, it will ultimately lead to serious heart failure. On the other hand, when individuals with fibrillation are able to undertake the work equal to that done by a perfectly healthy man, there is proof of such a degree of healthy heart muscle and freedom from valvular or muscular embarrassment that a good prognosis can be given.

We meet, however, with such varying degrees of exhaustion that an estimate must be acquired of the amount, though it is impossible to describe with accuracy what that amount may be. Even with distinct limitation of the heart's power, as shown by the response to effort, the prognosis may still be favourable, so long as the patient lives within the limits of his powers, and can avoid such efforts as cause him distress or exhaustion.

In transient attacks of auricular fibrillation, the attacks usually tend to become more frequent, until auricular fibrillation becomes permanently established. The prognosis of such cases depends on the way the circulation is maintained, this to be estimated in the manner already described. Transient attacks may appear for a short period and then disappear entirely. In two of my patients I detected a transient attack of auricular fibrillation, and in one, twenty-four years later, it became permanent (Observation 10). From the recognition of such cases we can conclude that auricular fibrillation is not of necessity a sign of grave significance.

In conclusion it can be said that auricular fibrillation is not in itself a serious condition, nor does it indicate the presence of any grave forms of disease. It arises from a loss of control of a part of the genetic system, and if the other structures of the heart are free from disease the condition is without danger.

The recognition that danger lies in the uncontrolled a.-v. node embarrassing a damaged ventricle by stimulating it to excessive rapidity, and as we may be able to prevent this danger, the prognosis of this condition in many instances depends upon how the heart responds to treatment.

Sudden deaths may rarely occur from the detachment of clots which have formed in the auricle and have passed into the circulation and lodged in the brain, or from sudden failure of the output, either from feeble ventricular beats or from ventricular fibrillation. No sign exists which gives a warning of such occurrences.

CHAPTER XXXIV

Treatment of Auricular Fibrillation

General.
The use of digitalis.
Method of administration.
Standardization of drugs.
The employment of digitalis.
Sudden death from digitalis.
Treatment by quinidine.

General.—When any individual with heart failure presents himself for treatment, it may be taken for granted that he has been undertaking a greater amount of exertion than the heart has been capable of performing without undue exhaustion. Hence the exhaustion of the heart's strength has been brought about in the first place by overwork. It may be that, embarrassed by the auricular fibrillation, the amount of work has been small, as measured by what a healthy heart can perform. With this conception of the cause of heart failure, the first and obvious course to pursue is to ease the heart of some of its work. In doing this much discrimination is required, and a thorough inquiry into the patient's mode of life has to be made in order to find out what circumstances, such as overwork, sleeplessness, digestive trouble, other illness, may have provoked or aggravated the heart failure. These have to be attended to in every case, and relief may be at once afforded with the removal of the disturbing or exhausting cause.

The use of digitalis.—While it is important to attend to such circumstances in heart failure with auricular fibrillation, as in all other forms, there are circumstances in cases of heart failure with fibrillation which when appreciated help greatly, not only in the restoration of the heart's strength, but in the prevention of heart failure. The most important is the rate of the pulse and the presence of the small ineffective beats, not only when the patient is at rest, but when responding to effort.

It is seldom that I have been able to say that I have saved a patient from immediate peril by the use of drugs; but this I can say with confidence, that I have repeatedly seen patients in great distress and in evident peril of death relieved of suffering, removed rapidly from danger, restored to a condition of comparative health, and even rendered fit for work by the judicious use of digitalis. The manner of its application needs, however, very careful attention, for it is a drug that needs to be applied on certain definite lines, if full benefit

is to be obtained from its action. I think it necessary to insist upon this point, for the somewhat 'rule-of-thumb' methods of its use, so generally employed, fail to get the full amount of benefit which this drug is capable of bestowing.

To understand the action of digitalis, it is necessary to appreciate the manner in which heart failure progresses in cases of auricular fibrillation, and the way it is controlled by digitalis. It may be taken for granted that when a patient with auricular fibrillation has a pulse-rate, or, to be more accurate, a ventricular rate, of 90 beats per minute and over when at rest, or when the rate is greatly increased on effort, he will in course of time gradually lose strength, his heart will become more feeble, and the evidences of heart failure will become more severe. This process may be very gradual, but it is very sure. On the other hand, heart failure may set in rapidly, more especially when the heart's rate rises to 120 and 140 and over. The severity of the failure, however it is brought about, compels the patient to seek rest, and we generally find such patients in bed, sitting up and breathing in a laboured fashion, with considerable distress, the heart usually dilated and the face of a bluish tinge, and possibly with dropsy and enlargement of the liver. In all such cases the prompt administration of digitalis is urgently called for, and, if given in sufficient doses, relief may be obtained in a few days, the relief being accompanied by a remarkable slowing of the pulse-rate. When this is accomplished, or when there are other signs of a sufficiency, the digitalis should be stopped for a few days, and resumed in small doses when the rate begins to increase. The rate of the pulse should be watched, and the quantity sought for which keeps the rate moderate when effort is made. It is seldom advisable to keep the rate under 60 beats per minute, although in some cases the patient feels fittest when it is at a rate of about 50 beats per minute. In this, we must be guided by the patient's sensations, the pulse-rate, and the manner in which he responds to effort.

Even when patients suffer from only a moderate degree of heart failure, and are able to go about, it is well to place them under the influence of this drug if the pulse-rate is over 80 beats per minute when at rest, or increases unduly when effort is made. My usual procedure in such cases is to attend to any circumstance that may aggravate the heart failure, and then to give the patient digitalis until the pulse-rate is reduced. If the failure is of some severity, I put him to bed until the proper effect is obtained, but where it is less in degree I permit him to go about his affairs.

In all cases where the heart has been sufficiently reduced in rate, I find out the quantity of the drug that is necessary to keep the heart at the rate at which it can perform its work with the greatest efficiency. In doing this, the patient's sensations are of the greatest help, whether he is confined to bed or attending to his affairs. He readily appreciates the change in his response to effort, and some such symptom as a disagreeable action of the heart or breathlessness can be employed as an indication that the heart's strength is being exhausted. Once the patient understands the meaning

of these sensations, he is generally quick to perceive what digitalis does for him, and its administration can usually be left quite safely in his hands. On such lines, I have seen many people lead useful lives for long periods of years with no bad effects, except when they have not taken the drug in sufficient quantities to keep the heart at the required rate.

The foregoing line of treatment is applicable chiefly to cases in which auricular fibrillation has arisen recently, or where the heart failure is of recent date. In more advanced cases, when the condition has induced from time to time periods of heart failure, and there has appeared the change that accompanies chronic heart disease, such as persistent shortness of breath, enlarged liver, and dropsy more or less continuous, the persistent use of digitalis may still tend to restore a measure of strength to the heart and give relief, enabling the individual to lead a useful life, though at a lower level, for an indefinite period.

In the search for an appropriate line of treatment in old-standing cases I have used many methods and many drugs, often with little or no benefit, but in a certain proportion of apparently hopeless cases I have seen extraordinarily good results following the use of digitalis, pushed until a reaction was obtained, and then stopped for a time, and again resumed, time after time. Not infrequently, after it has seemed useless to continue the drug, I have seen the individual acquire such an amount of strength as would scarcely have been anticipated.

As the conditions preceding auricular fibrillation and producing it are often accompanied by changes in the heart muscle of a slowly progressive nature, it is easy to recognize that the heart's strength cannot always be restored, and that as the amount of efficient muscle becomes reduced, a period is reached when no method of treatment is of avail.

Method of administration.—A great diversity of opinion is to be found in regard to the form in which digitalis should be given, and also in regard to the dosage. So far as I have worked out the subject in regard to auricular fibrillation, I see no reason for giving the preference to any particular preparation. The best and most assured way, in cases of marked failure, is steadily to push the drug, whichever form be employed, until a reaction is observed. Usually the digestive system is the first affected, loss of appetite, nausea, vomiting, or diarrhœa being set up, the patient usually feeling ill and miserable. If the digitalis is effective on the heart, as a rule a marked slowing of the pulse is found at the same time, or even before any digestive disturbances arise. If there is dropsy, the first sign of digitalis action is sometimes a great increase in the flow of urine. In some cases, a slowing of the pulse is the first sign of a sufficiency. When this stage is reached, the administration of the drug should be stopped for a few days. In a day or two, after stopping the drug, patients feel remarkably well and bright, and if nausea was present, it disappears. The heart-rate is carefully observed, and when the rate shows signs of increasing, half the amount which was previously found effective should be given, and the dose increased or diminished according

to the manner in which it affects the rate, the object in view being to give just the amount which enables the heart to carry on its work with the greatest efficiency. As previously stated, the patient himself by his own sensations speedily acquires the knowledge of how much of the drug is needed, and by attending to his own experiences, he will soon find out the smallest dose which is needed to give the best results.

A good deal of my work has been done with the tincture of digitalis; and I have used this preparation for over forty years, and have never yet come across an ineffective preparation, my standard being the reaction in susceptible individuals.

The quantity I usually start with, where the failure is marked, is 1 drachm of the tincture per day, in doses of 15 to 20 minims. This is steadily pushed until a reaction is obtained; then it is stopped and employed in the manner already described. Usually a reaction is obtained within a week, sometimes in a few days. Where there is great distress and more urgency, I give as much as 2 drachms of the tincture daily, and then get a reaction in two or three days.

I have frequently used Nativelle's digitalin granules, and find them also very efficacious. I have found that one of these granules, containing $\frac{1}{240}$ grain of digitalin, is equal to 15 minims of the tincture.

Other drugs, such as strophanthus and squills, have the same effect as the digitalis, and in some cases they may cause less digestive disturbance; but in the majority of cases I have found that when the digitalis is ineffective, so also are these drugs. In many cases the effects of digitalis are less disagreeable than these other drugs.

In some urgent cases it may be necessary to produce a reaction more speedily, though I have rarely failed to get a reaction in good time by giving digitalis by the mouth. In order to obtain a speedy reaction, strophanthin or strophanthone may be injected into the veins. In a series of observations which have been carried out at the Mount Vernon Hospital and at the London Hospital, it has been found that in auricular fibrillation with a pulse-rate of over 140 per minute, intravenous injections of strophanthin ($\frac{1}{250}$ gr.) can reduce the rate and give relief in five to eight hours, but I am of opinion that it is only in very exceptional and urgent cases that this method is required.

The standardization of drugs.—Of late years there has arisen a desire to obtain remedies so effective that their potency can be relied upon. But not only has the desire been to obtain remedies of a standardized potency, but there has also been a desire to administer them to patients upon some standardized principle. Such a desire is highly commendable, for the dosages recommended in the different pharmacopœias are so vaguely described that it is not known how they should be administered in order to obtain their full benefit.

While by means of chemical testing and by experiment upon living animals the potency of a remedy can be accurately measured, it is a totally different matter to measure the susceptibility of the patient to the remedy. We find elaborate calculations of the quantity of sera, or of a drug like digitalis, which

should be administered to patients, the body weight being taken usually as a standard. Such a procedure shows that those who recommend it have failed to grasp the principles which lie at the foundation of therapeutics. Drugs act in the same manner and on the same structures as do the agents of disease. It is manifest, therefore, that the action of a drug on a healthy animal may have a very different reaction upon an individual affected by disease. Body weight is no criterion. A small dose of castor oil may have a powerful effect upon a man 16 stone in weight, while it may have no effect upon a woman who weighs but 7 or 8 stone.

But of all drugs given according to body weight, drugs of the digitalis group are those which should never be administered in this way. The reason for this has been commented upon in the discussion of auricular fibrillation and auricular flutter, where the great susceptibility in these conditions was shown and compared with the insusceptibility when the rhythm was normal. If this difference has been grasped, the futility of such a procedure becomes manifest. The two diagrams (Figs. 123 and 124) bring this out. Fig. 123 shows an individual example, while Fig. 124 shows the difference of the effect in normal hearts and in hearts with auricular fibrillation. Digitalis should be administered in quantities that are known to produce definite effects when sufficient is administered. It behoves the doctor, therefore, to recognize these quantities, and to recognize the symptoms which a sufficiency of the drug produces, and to push the drug courageously until there are evidences of its efficiency. The pushing of the drug till evidence is obtained of its efficiency is one of those principles which should be the guide in the use of every remedy.

The employment of digitalis.—It is extremely difficult to get the members of the profession—teachers, heart specialists, general practitioners—to understand how to administer digitalis, and I repeat again some further points. In the treatment of patients with auricular fibrillation by means of digitalis a great deal of judgment and intelligence has to be used. There is no rule of thumb, for every patient has to have the treatment modified to suit his special requirements. It is necessary to recognize this, for the real principles have not been grasped. I have seen many patients gravely ill, patients who were getting digitalis, but receiving insufficient doses. I have seen others sick and ill and their lives imperilled by too large doses. The presence of auricular fibrillation is not necessarily an indication for digitalis, for many patients require no treatment whatever. The great guide is always the patient's sensations, and particularly those which occur in response to effort. Many a time the patient knows better than the doctor when he wants the drug and when he has had enough. This lesson I learned from such patients as that described in Observation 6, his peculiar test being his inability to lace his boots on account of the œdema of his feet. Most patients recognize their need for the drug by increasing breathlessness.

It must also be remembered that with patients who benefit by the use of digitalis there will come a time when they can do perfectly well without it. This is due to the a.-v. node having become less excitable, so the ventricle

does not run off when effort is made. Another point which has to be insisted upon is that in giving digitalis we must not be content only to give so much as to reduce the pulse-rate when the patient is at rest. It is necessary to see that the rate does not become excessive when effort is made. I was astonished to see lately in a journal the statement made that 'digitalis in ordinary therapeutic doses fails to protect the ventricles from the exaggerated

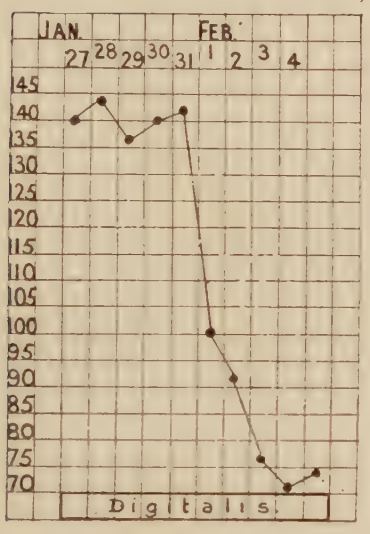


Fig. 123. Chart showing a typical reaction of digitalis in a case of auricular fibrillation with severe heart failure. The administration of the tincture of digitalis began on January 27, in doses of 15 minims four times daily.

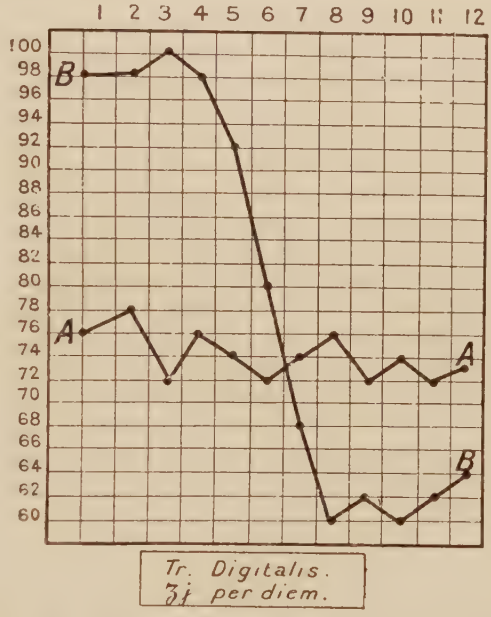


Fig. 124. Chart showing the effects of similar doses of tincture of digitalis on six cases of mitral stenosis with the normal rhythm (*A*) and six cases of mitral stenosis with auricular fibrillation (*B*). The figures at the side represent pulse-rate; those at the top, days. In each case the digitalis was continued till the heart became slowed, or until nausea or vomiting occurred. The average quantity before an effect was produced was 7 drachms (1 drachm per diem). This had little or no effect on the heart-rate in the patients with the normal rhythm (*A*), while there was a rapid decrease in the rate in the patients with auricular fibrillation (*B*).

response to effort'. It is manifest that the writer had never given his patients a sufficient quantity of the drug. I had thought that I had made this quite clear many years ago. Here is an observation which is typical of many.

OBSERVATION 20.—A man, aged 37, consulted me in 1914. He suffered from mitral stenosis, and had auricular fibrillation with a rate that varied from 70 to 100 beats per minute when at rest. On moderate effort the rate speedily rose sometimes between 120 and 130 beats per minute. He was put on digitalis, and the drug was steadily pushed till the rate fell to about 60 and sometimes under. The response to the same amount of effort produced a much less

increased rate—the rate rarely rising above 85 beats per minute. The details are brought out in the diagram (Fig. 125) which Dr. John Parkinson has made in this case. Under the treatment by digitalis this man has led a vigorous and active life, and is to-day (ten years later) still capable of a good deal of bodily effort.

The same features were brought out in another case, which is represented in the diagram (Fig. 126).

I have been called in consultation a great many times to see patients whose condition had been recognized, and to whom digitalis had been

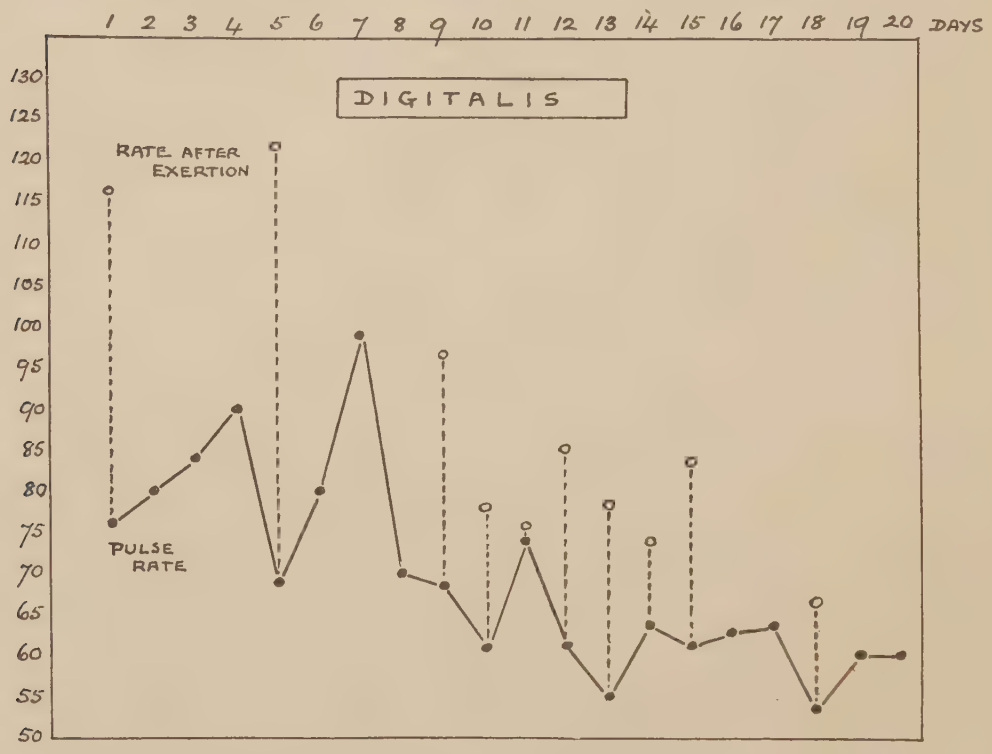


FIG. 125. Chart showing the effect of digitalis in preventing an excess in the increase in rate after effort. Before digitalis a measured amount of effort raised the pulse-rate from 75 to 115, and from 65 to 120. After digitalis, which was taken from the 6th to the 13th day, the rate slowed when at rest, but the same amount of effort resulted in a much less increase in rate; the last response showed that effort raised the pulse-rate from 54 to 66.

prescribed by teachers of medicine, and little benefit had accrued because of the insufficient dose.

OBSERVATION 21.—A few months ago a man, 65 years of age, came to St. Andrews on a visit. He suffered so much from his breathing that the hotel proprietor strongly urged him to see me. His story was very instructive. He was well and vigorous up to four months ago, after which he noticed that he had been gradually less able to undertake effort because of breathlessness. His doctor called in consultation a professor of medicine, who diagnosed auricular fibrillation and prescribed digitalis, which had been continued ever since. In spite of this and

rest in bed he made no improvement. When I saw him his pulse-rate was about 70 per minute when resting, but went up to over 120 on slight effort. He was unable to walk much because of the breathlessness, and he had to be propped up in bed because of the dyspnœa. The quantity of drug he had been taking was 5 minims of the tincture three times a day. I told him to take 30 minims three times a day, and to call again in four days' time. A different man presented himself in four days. He came into my consulting room looking alert. He stated he felt as well

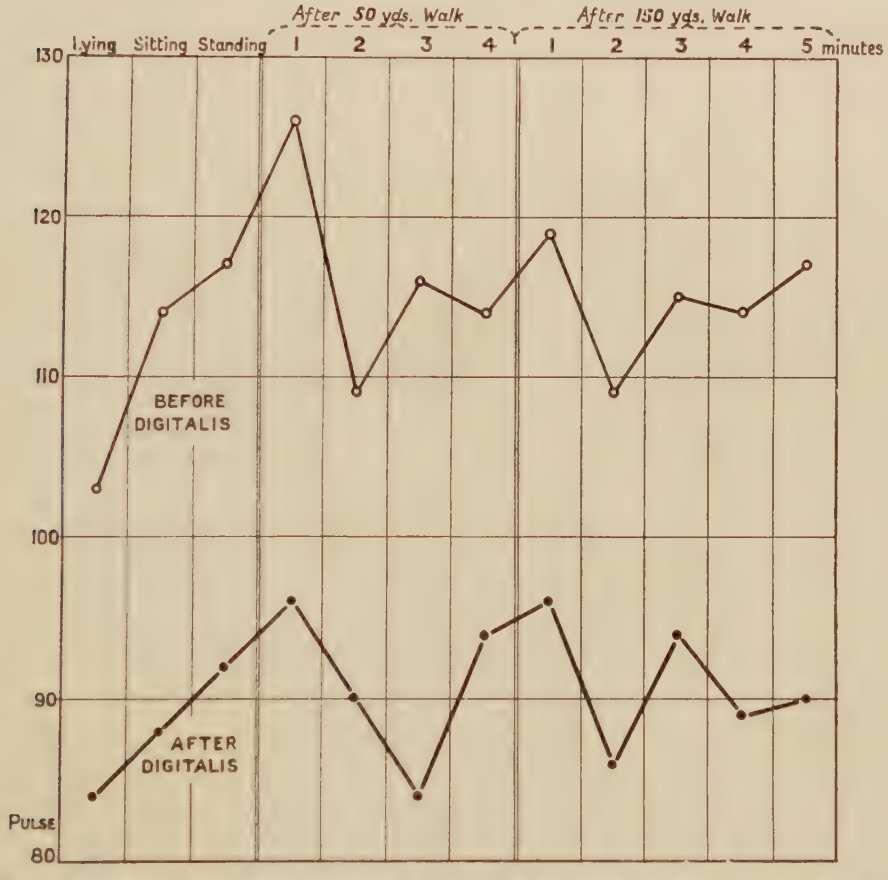


Fig. 126. Chart showing the control of pulse-rate on exertion, after 9 drachms of tincture of digitalis in nine days. The upper record was taken before digitalis, and shows a varying rate reached when lying, sitting, standing, after walking 50 yards, and after walking 150 yards. The same test was applied after the digitalis, and is shown in the lower record.

as ever. The previous night he had slept for ten hours with only one pillow, a thing which he had not done for three months. He could walk long distances without the slightest distress, The pulse-rate was about 65 when resting. The same effort which previously caused the pulse-rate to rise to 120 beats did not now raise it above 80 beats per minute.

Sudden death from digitalis.—There has been for many years a vague notion that digitalis was a drug that was capable of causing sudden death. Indeed, one famous therapeutic authority declared that when a patient was

taking digitalis he should be kept in bed, and he cited an experience in which a patient under digitalis on getting up to the stool fell down dead. For many years this question of sudden death from digitalis has interested me greatly. I have had practically no experience myself. The only occasion when it might have occurred was a man whom I was treating shortly after I recognized ' auricular paralysis ' as a clinical entity. Digitalis had no effect upon this patient's heart. The rate was not very great—about 80—and I was carefully pushing the drug when he died suddenly.

For many years I have prescribed digitalis to all and sundry, and pushed the drug until I got a reaction that could be attributed to the drug, such as nausea and vomiting, or marked slowing of the pulse, or partial heart-block. In some cases large quantities were given, for I had found that with the same preparation the quantities that produced the result varied greatly, but I never had another death which could be attributed to the drug. This freedom from such a result I attributed to the fact that the drug was stopped whenever the patient showed any kind of reaction.

Whenever I heard that a patient had died suddenly when using digitalis I contrived to get into communication with the doctor, and by inquiring into the patient's condition I invariably found that the pulse was irregular and that the rate had become slow, and frequently with a coupling of the beats, so I have come to the conclusion that the danger from digitalis only occurs in auricular fibrillation.

A few references in literature point in the same direction. It is a pity that observers are so chary of recording their failures, for we would learn much more from these than from the glowing accounts of their successes. Here is one record which I have found. In his *Lectures on Pathology*, published in 1891, p. 384, H. G. Sutton says, ' The difficulty in using digitalis is to know how much to slow the heart '. He goes on to describe patients with an irregular rhythm to whom digitalis was given in large doses ; the rhythm becomes more regular and less rapid. ' The drug was slowing down the heart nicely,' says the house physician. ' Oh, Doctor, I feel so much better,' says the patient. In a few hours the patient was found dead. Sir William Gull said to Sutton, ' Sir Thomas Watson and I were treating a case of dilated heart. We gave digitalis. At the next visit we found the heart much more regular, and we were leaving the house feeling very satisfied when suddenly the patient died.'

I was called to see a man said to be dying from heart disease. He had to sit up in bed and breathed heavily ; his face was livid. He had dropsy, an enlarged liver, and a large and irregular heart beating at the rate of 130 to 140 per minute (auricular fibrillation). I told his doctor to push the digitalis till he showed evidences of a sufficiency, either by the slowing of the heart or nausea, and then to stop it. After five days he telephoned me that the patient was wonderfully free from distress, could lie flat, had a good colour, and the dropsy had almost gone, the pulse-rate being between 70 and 80. I told him to stop the digitalis for a few days, and if the pulse

then increased to give smaller doses, and find out the exact quantity which kept the rate about 80. Three days later he telephoned to say that the patient had been going on well, but that morning, during the doctor's visit, the patient fell back and died. I asked the doctor if he had stopped the digitalis, and he replied in the negative, saying that, as it had done him such a lot of good, he had continued it, in spite of my directions.

Treatment by quinidine.—During the past six years my endeavours have been directed mainly to finding out the nature of symptoms, and I have not had the opportunity of personally watching the effects of quinidine. From all reports it is proving itself to be a very potent drug, potent both for good and for evil, and it evidently requires great discrimination in its use. While it seems to have the power of restoring the normal rhythm in a goodly proportion of cases, its use is not without danger. One source of danger was perfectly clear to me when I read the first account of its action, viz. the throwing out of fragments of clot which may have been formed in the auricle after the onset of auricular fibrillation. In some patients with auricular fibrillation numerous clots may be discharged and lodged in different arteries. On a few occasions this has happened with the restoration of the normal rhythm where auricular flutter had been present. The possibility of its occurrence makes me always somewhat anxious when pushing the digitalis to restore the normal rhythm in auricular flutter. This result has happened now in a considerable number of cases where quinidine had restored the normal rhythm in auricular fibrillation.

It is, as I said, unfortunate that observers do not faithfully chronicle their failures and disasters. While a good case can be made out for the use of this drug, there are incidents of which one hears, and which an occasional writer faithfully describes, where attacks of an alarming nature with collapse and loss of consciousness and sudden death occurred. Another matter which requires consideration is the fact that in a goodly proportion of cases the recovery is but temporary. Any one with much experience of auricular fibrillation will recognize that this is to be expected, for it is a common experience for patients to have, it may be for years, temporary attacks of fibrillation before the condition becomes permanently established. If, as I anticipate will be found to be the case, auricular fibrillation is often the consequence of fibrotic degeneration of the s.-a. node and the auricular portion of the genetic system, it will be realized how unlikely it is that a permanent recovery can take place.

Another point to be borne in mind is that auricular fibrillation is not in itself necessarily a dangerous condition, and that its worst effects occur where there is already extensive damage to the heart muscle. The danger arises from the rapid action of the ventricle and the small and imperfect beats. In the vast majority of cases this danger can be obviated by the judicious use of digitalis.

I mention these points not in any way to prevent the use of the remedy, but that the remedy should be used with discretion, and an endeavour made to recognize the cases likely to benefit, and the cases likely to be rendered

worse. It must always be borne in mind that its action is still a matter of guess-work, notwithstanding the attempts that have been made to explain its action. We know far too little of the factors concerned in regulating the action of the normal heart, and of the conditions which lead to these abnormal rhythms, to be able to recognize the effect of remedies. We have just to reflect upon the enormous amount of work that has been spent upon investigating the action of digitalis, and to recognize that we are still just beginning to appreciate its action, to realize that a great deal yet has to be learned before we can understand the action of quinidine.

CHAPTER XXXV

Auricular Flutter

The recognition of auricular flutter.
The diagnosis of auricular flutter.
The patient's complaint.
The inspection of the jugular pulse in auricular flutter.
The diagnostic character of the radial pulse in auricular flutter.
The pulsus alternans and irregular pulse in auricular flutter.
The jugular pulse.
The electro-cardiogram.

The recognition of auricular flutter.—There is another condition so closely allied to auricular fibrillation that it has in all probability the same origin. This is a condition which MacWilliam called ' flutter '. The auricles contract at a rate varying, as a rule, from 250 to 300 beats per minute. MacWilliam first described this condition as a result of experimental stimulation in 1887. In 1905, W. T. Ritchie obtained graphic records in a patient where the auricles were beating at 274 per minute. In 1908, Jolly and Ritchie obtained electro-cardiographic records of the fluttering auricles from Ritchie's case. When Ritchie called attention to this condition, I turned to my records and recognized a number which showed this condition of flutter, but which I had interpreted as being of the same nature as auricular paralysis, a term that I then applied to auricular fibrillation. Amongst my records I found instances where flutter and fibrillation alternated, and as years went on, in many of my patients, auricular flutter passed into auricular fibrillation.

When auricular flutter was recognized as a clinical entity distinct from auricular fibrillation, I tried to obtain the means of recognizing it by the unaided educated senses, as I had done in the case of other irregularities. But the phenomena, particularly those revealed by the pulse, were so protean that it seemed likely that for the diagnosis of this condition mechanical devices would always be necessary for its recognition. In later years I renewed the attack and reviewed the whole of my observations, and I have found that from being the most obscure of abnormal rhythms it is among the easiest to recognize without the aid of instruments.

The diagnosis of auricular flutter.—The chief points to be noted in the diagnosis are : first, a clear account from the patient of the onset of the abnormal heart action when the condition is transient, the offset of an attack,

and the behaviour of the heart during the attacks, as to the rate and rhythm ; second, the inspection of the jugular pulse, for the rapid movements which are produced by the fluttering auricle ; and third, the peculiarities in the rate and rhythm of the pulse that may be brought about during periods of rest and effort.

The patient's complaint.—The patient usually gives a history of heart attacks which begin suddenly and stop suddenly, and which last for varying periods of time, of a few seconds, minutes, hours, or days. Such patients recognize the onset by a rapid and regular action of the heart or by a curious fluttering sensation within the chest, often accompanied by a sense of prostration, sometimes extreme. Some have attacks of unconsciousness, lasting from a few seconds to hours.

The story of heart attacks during which the heart beats rapidly but regularly should always raise the presumption of auricular flutter, particularly if the attack ends suddenly with one or two forcible beats. If the attack persists for hours or days, different people show different degrees of heart failure. In some there is only the sense of weakness which limits their efforts, in others there is breathlessness with the onset and all degrees of heart failure of that kind due to deficient output of blood, such as those already described as following the onset of auricular fibrillation.

With the resumption of the normal rhythm all the symptoms of heart failure speedily disappear.

These symptoms are those experienced by the patient during attacks of paroxysmal tachycardia from causes other than flutter. (See Chapter XXVI.) In my experience the attacks from flutter are far more frequent.

The inspection of the jugular pulse in auricular flutter.—It often happens that the diagnosis is at once made by inspecting the movements of the veins in the neck. These movements are usually from 250 to 300 per minute, and quite regular, and when present there is nothing more diagnostic. The waves are sometimes large (as in Fig. 127), and at other times small and barely perceptible (Fig. 128), and then only to be found by careful inspection of the neck immediately above the inner end of the right clavicle where the jugular bulb is situated. On a few occasions I have found but one large wave in the jugular, synchronous with the systole of the ventricle. This type of jugular pulse will be referred to in dealing with the graphic records.

The diagnostic character of the radial pulse in auricular flutter.—In no other condition are the rate and rhythm and size of the pulse so varied as in auricular flutter. At first sight it seems impossible to give any directions which would help in discriminating the pulse abnormalities of this condition, and for many years I puzzled to find a method of recognizing auricular flutter without the use of instruments. My records from patients with auricular flutter exhibited such a large variety of phenomena that I was in despair of obtaining a clear method by which the condition could be recognized by the unaided senses. Auricular fibrillation, on the other hand, was comparatively

easy to recognize, but flutter baffled me until I applied the principle of the reflex arc to the interpretation of the phenomena.

Briefly, auricular flutter can be recognized by the fact that, if the pulse is in the neighbourhood of 150 per minute and regular, no amount of effort

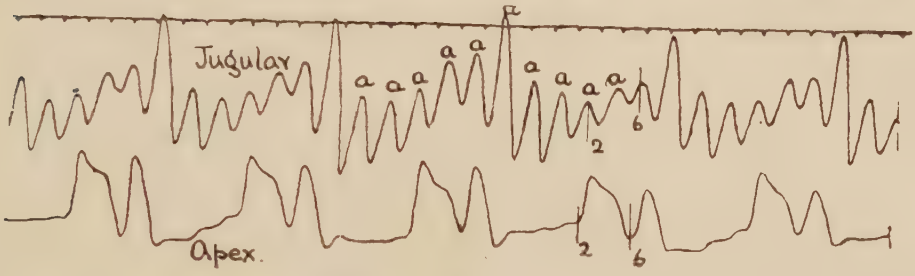


FIG. 127. Simultaneous tracings of the jugular pulse and apex beat. The auricular beats (*a*) are 250 per minute, and the ventricular 60.

will increase its rate. In only one patient did the rate increase, and then it became doubled, being nearly 300 beats per minute, and then it suddenly fell back to 150. That is to say, there was no intermediate rate between the 300 and the 150, the ventricle either responded to all of the auricular impulses or to half. When the pulse-rate falls below the 150 it at once becomes irregular, as shown in Figs. 7, 8, and 9. Occasionally it may be regular, but that would be when the ventricle responded to every three or

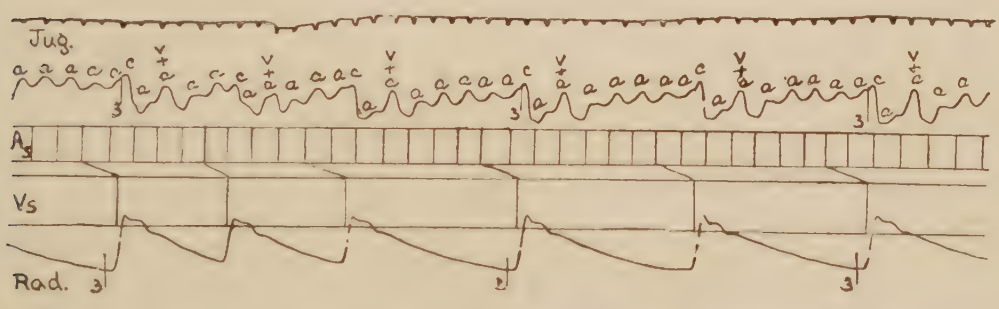


FIG. 128. Shows an auricular rate (*a*) of 220. The radial pulse is irregular, due to the ventricle responding to a varying number of auricular impulses, as shown by the intercalated diagram.

four auricular impulses. But here again any increase or decrease in the rate resulted in the pulse becoming irregular.

The explanation of this peculiar behaviour of the pulse is given with the description of Figs. 7, 8, and 9 on pp. 45 and 46.

So that, if we find a patient whose pulse is about 150 and effort does not increase it, then one may recognize that it is a case of auricular flutter. Or if it falls below 150 and at once becomes irregular, this is another evidence

of auricular flutter. It will be observed that there is a marked difference in this from any other known condition. In people with a normal acting heart, the rate varies with effort, and in its variation the rhythm is never irregular.

The pulsus alternans and irregular pulse in auricular flutter.—The pulsus alternans is frequently present in auricular flutter when the rhythm is regular and the rate about 150 (Fig. 7) or above double that number (Fig. 135). Its presence alone is an almost certain indication of flutter. The origin of the disturbance that leads to the pulsus alternans has been the subject of a good deal of discussion, and the matter is still far from being settled, and new explanations keep cropping up. It seems that these tracings afford a new explanation as it occurs in auricular flutter. In Fig. 7, where a small beat alternates with a large, the alternation seems to be due to the varying activity of the a.-v. node—which is the explanation given in the book for

Fig. 129. Tracing of the jugular and radial pulses during an attack of auricular flutter, as revealed by the electro-cardiogram (see Fig. 130). The venous pulse is of the ventricular form.

the small ineffective beats in auricular fibrillation. This point will be touched upon again in the chapter on the pulsus alternans.

The jugular pulse.—There is a remarkable difference in the size of the waves which one observes in the jugular pulse, as can be inferred from the records of Figs. 127 and 128. The waves in the patient who gave Fig. 127 were visible at a distance of several yards. In many cases they are too small to give a record and they can only be perceived by the sight. It seems that the size of the wave may have some relation to the condition of the auricle and, it may be, to the degree of dilatation.

In a few instances I have found that the jugular pulse in auricular flutter was due, not to the auricle but to the ventricle. This is recognized by the jugular pulse being synchronous with the radial. It is then of the ventricular type and is of exactly the same kind which occurs in auricular fibrillation. When the pulse is irregular it is indistinguishable from that of auricular fibrillation. This is shown in Fig. 129, which at first sight would be taken as diagnostic of auricular fibrillation, but an electro-cardiogram shows that auricular flutter was present (Fig. 130).

When this ventricular venous pulse is quite regular and attains a rate of about 150 per minute, we can safely assume that auricular flutter is present (Fig. 139).

The ventricular venous pulse in these cases seems to be due to free tricuspid

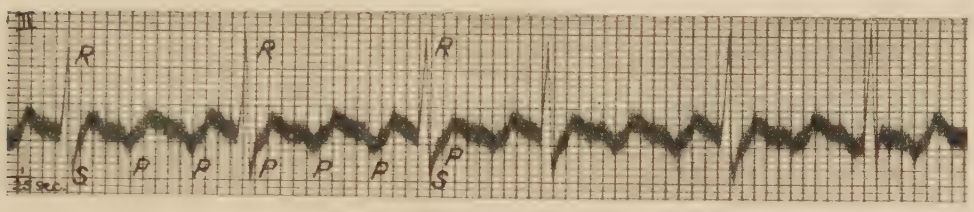


Fig. 130. Electro-cardiogram taken during the same attack of tachycardia, as shown in Fig. 129. The auricular movements (*P*) are 310 per minute, and the ventricular (*R*) 120.

regurgitation, and the auricle is so dilated that the movements due to the flutter produced no effect on the contraction of the auricle.

It will be noticed that in the jugular tracing in Figs. 131-136 the waves alternate in size. This is due to the auricular contraction occurring at one time when the ventricle is in systole, when no blood can flow into the

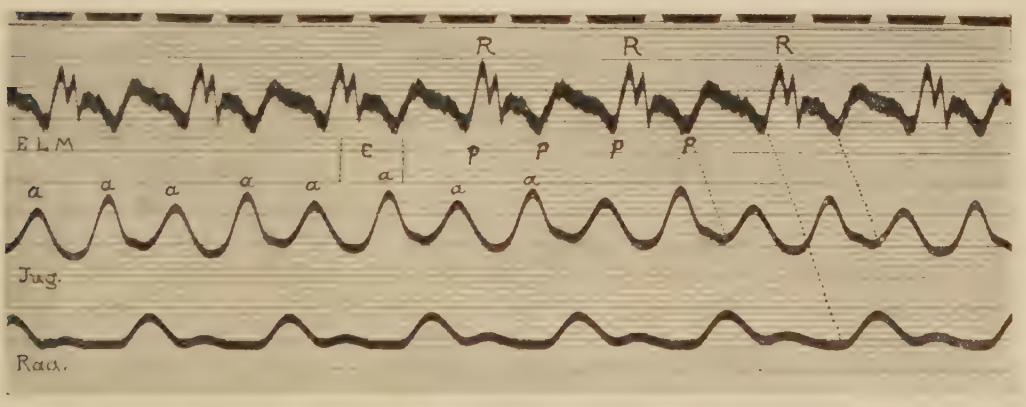


Fig. 131. Electro-cardiogram (*E.L.M.*) and graphic records of the jugular (*Jug.*) and radial (*Rad.*) pulses from a patient with auricular flutter, to show the nature of the alternation in the size of the jugular pulse. The auricular beats (*P* and *a*) are twice the rate of the ventricular, and one auricular beat falls during ventricular systole (space *E*) and the other during diastole. In the latter case most of the contents of the auricle pass into the ventricle, while in the former case none can enter the ventricle, and so the contents are forced back into the veins, giving rise to the larger beat. (J. Parkinson.)

ventricle, so that all the blood is forced back into the veins, thus giving rise to the larger waves. At the other time the auricular contraction occurs during ventricular diastole, and a portion of the blood flows into the ventricle, giving rise to the smaller waves.

These points are well brought out in Fig. 131. On one occasion we obtained the waves of flutter of the left auricle from the apex tracing, Fig. 132.

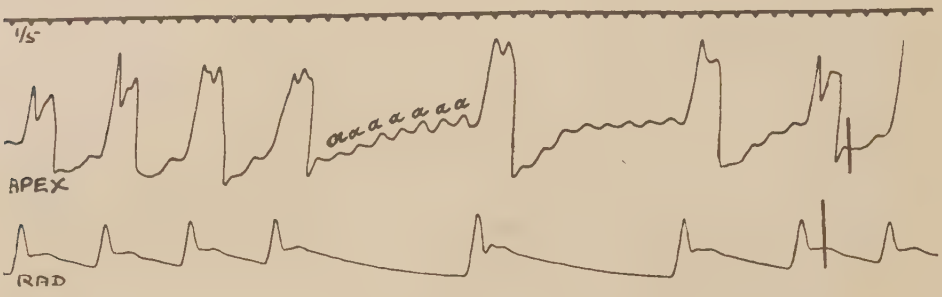


Fig. 132. Tracings of the apex beat and the radial during auricular flutter, after digitalis had slowed the ventricle. The waves (*a*) are the auricular movements affecting the apex of the left ventricle.

The electro-cardiogram.—The electro-cardiogram of flutter is characteristic, showing a regular sequence of waves from certain of the leads (Figs. 130 and 131). From other leads there may be no sign of auricular activity (Fig. 133).

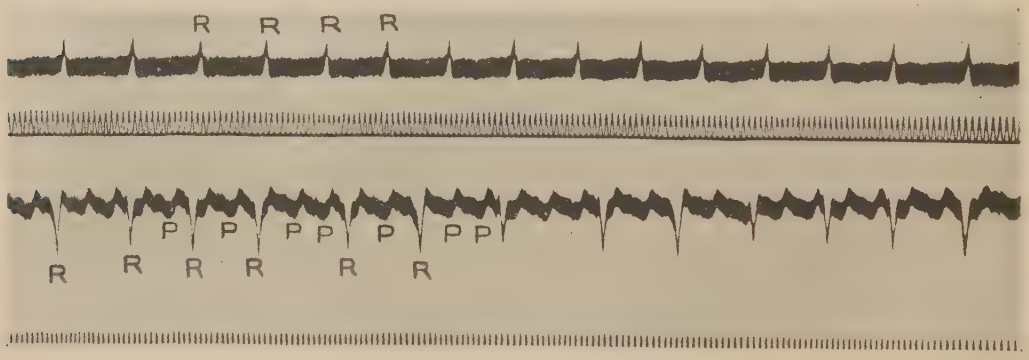


Fig. 133. Upper record is by derivation I.; lower record is by derivation III. The record from derivation I. shows no waves due to the auricle. They are shown in derivation III. *P* waves. Time-record in each is that of a tuning-fork, 28·57 to a second. Auricular flutter. Auricular rate = 302 per minute, with irregular ventricular responses.

CHAPTER XXXVI

Cases illustrating Auricular Flutter

Observations 22-36.

OBSERVATION 22.—A man who consulted me in 1913, at the age of 52, stated that from the age of 15 or 16 he was conscious of short attacks of rapid action of the heart. Notwithstanding these, he worked hard as a clergyman until 1906, when, during an attack, he lost consciousness. After some months' rest the attacks ceased. In 1909 he noticed that he was becoming gradually weak and short of breath, and the doctor found his pulse irregular, the rate being 130 per minute. In spite of treatment this rapid rate persisted till I saw him in 1913. An electro-cardiogram then revealed that the rate of the auricle was 300, and that of the ventricle 150 per minute. While under my observation, the rate would sometimes fall to 130 and under, and become irregular. In spite of treatment by big doses of digitalis, the auricle was unaffected, although sometimes the ventricular rate fell as low as 74 per minute, being irregular.

Since that date the patient has led a quiet life, the flutter still persisting, but the pulse, on different examinations, varying from 70 to 130, with short runs of regular beats. He is still, at the age of 63, able to get about in a quiet way, only he is short of breath and easily tired.

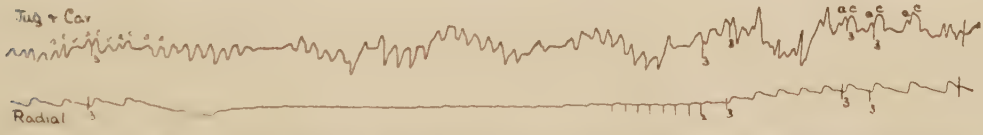


Fig. 134. Tracings taken during a short attack of auricular flutter in 1914, during which the patient lost consciousness. From the character of the radial tracing it can be inferred that the ventricle contracted at a rate of 200, but the output was so small as barely to affect the radial pulse (Theodore Thompson). In 1921, permanent auricular fibrillation set in and the attacks of loss of consciousness ceased.

OBSERVATION 23.—Another patient stated that he had had attacks of rapid heart action from the age of 18, and that sometimes he would become unconscious for a brief period (Fig. 134). When I saw him in 1913 he was having these attacks rather frequently, and an electro-cardiogram, taken during an attack, showed that they were of the nature of auricular flutter. I could do nothing to prevent their occurrence; the attacks came on unexpectedly, sometimes two and three in a week, and sometimes for months he was quite free. After this he consulted many doctors, and in 1916, while undergoing a course of treatment, the attacks ceased. I saw him in 1920, and he told me of the marvellous cure that had been effected, and that he had had no return of the attack for four years. On examination I found that he had auricular fibrillation, the rate being moderate. He is still, at the age of 59, leading a fairly active life.

OBSERVATION 24.—A patient whom I saw in 1910, aged 47, stated that he had had attacks of palpitation since 1901, after recovery from a mild attack of typhoid fever while he was serving

in the South African War. The attacks occurred at times with great frequency until 1903, when they produced loss of consciousness. In 1906 the attacks were very severe, but after two months' rest they became less severe and less frequent. In 1910, when he came under my care, the attacks

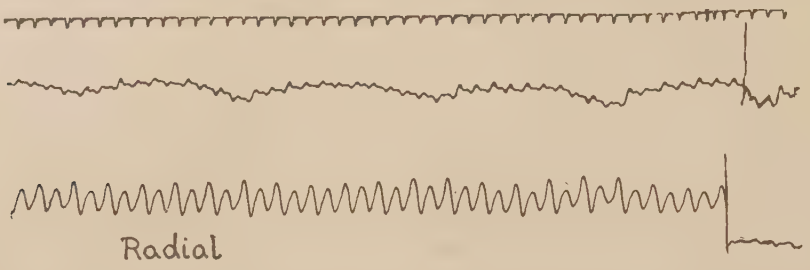


Fig. 135. Tracing of the radial pulse, and of the pulsation in the neck (probably carotid) during an attack of auricular flutter. The electro-cardiogram revealed an auricular rate of 290-300 per minute, with the ventricular contractions at half this rate. Occasionally for short periods the ventricular rate equalled the auricular, and during one such period this tracing was taken. Note the pulsus alternans in the radial tracing (Observation 24).

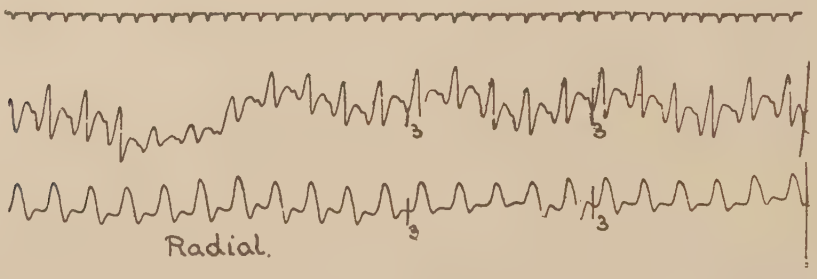


Fig. 136. Shows a period of auricular flutter with radial pulse-rate 140 per minute, auricular rate 280.

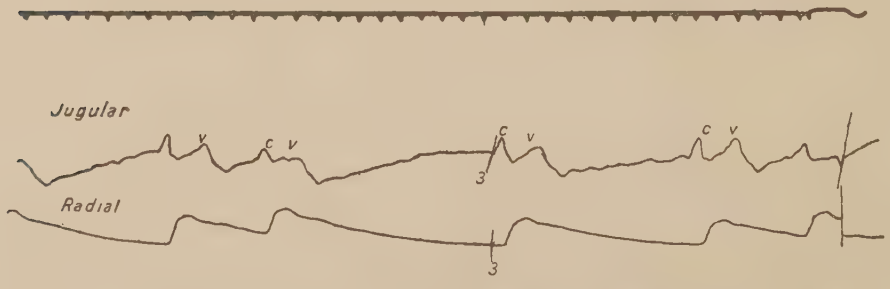


Fig. 137. Shows the presence of auricular fibrillation, pulse-rate 45 per minute as a result of digitalis.

were fairly frequent, lasting for weeks. Sometimes during this period the pulse, which was usually about 150, would suddenly rise to nearly 300 beats per minute (Fig. 135). Such attacks might last from half an hour to an hour, the patient being in great distress, chiefly on account of extreme prostration and the feeling that he would die. Digitalis produced in this patient the effects shown in Figs. 136, 137, 138.

When the heart rhythm became normal in response to the digitalis, as shown in Fig. 138, it for seven days kept reverting to fibrillation, as in Fig. 137. He gradually improved, though he suffered from short attacks of flutter lasting only a few minutes. In 1914 I found the auricles had passed into fibrillation, and digitalis kept the rate in check. Since that time he has had no more paroxysmal attacks, but the heart rhythm is permanently irregular, and he goes long periods without digitalis. In 1924, at the age of 61, he is able to get about in a quiet way.

OBSERVATION 25.—In 1911 I was called to see a lady 53 years of age, suffering from extreme heart failure. She lay propped up in bed, unable to lie down because of breathlessness. Cheyne-Stokes breathing was present. Her face was slightly livid, and the lips blue, and there was extensive dropsy of the legs. The pulse was very small and rapid, but the jugular pulse was large, regular, of the ventricular type, 150 per minute (Fig. 139). She had gradually drifted into this condition, which had continued for six weeks, in spite of various remedies recommended

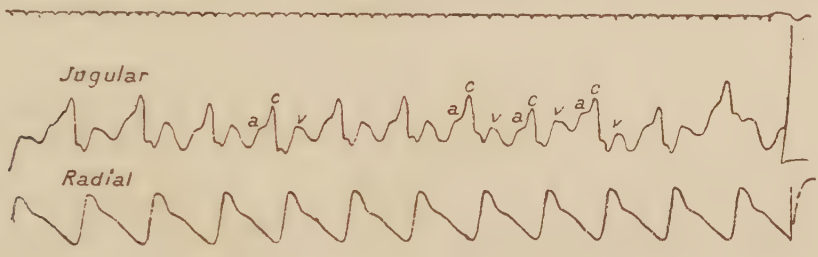


FIG. 138. Shows a normal rhythm established.

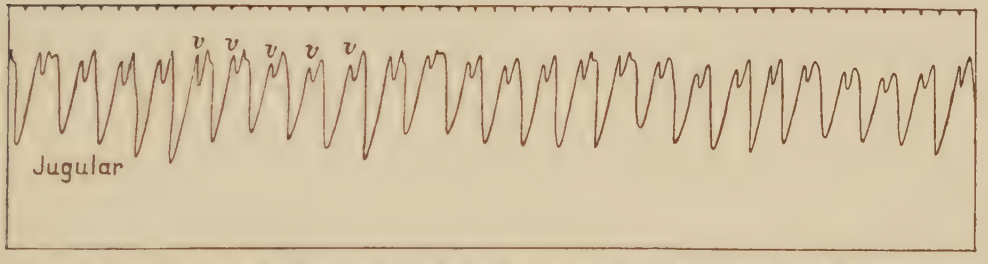


FIG. 139. Tracing of the jugular pulse of the ventricular type due to auricular flutter, taken in 1911.

by her doctor and sundry consultants. When I recommended digitalis the doctor pooh-poohed the suggestion, saying that it had already been given. When I asked in what quantity it had been given, and found that only 10 drops of the tincture three times a day had been given, I insisted that she should have 30 drops three times a day for four or five days. The heart speedily responded, and in two days she was able to sleep and lie down, and all the symptoms of heart failure disappeared within a week. I saw her again a year later. On examination I found the pulse irregular, and an electro-cardiogram showed that the auricles were fibrillating (Fig. 140). She was readily influenced by the digitalis, and to-day, in 1924, at the age of 66, she is able to lead a fairly active life, though the auricular fibrillation still persists.

OBSERVATION 26.—Male. Aged 74. He was well until January 30, 1910, when he noticed his heart was beating very irregularly, and after this he felt weak and was easily rendered short of breath. The rapid action of the heart persisted up to the time that I saw him in May 1910. So long as he kept quiet he felt no distress, but he felt weak and disinclined to make any exertion. He lay in bed, slightly propped up, his face and lips being of a dusky hue. The veins of the neck were distended and beat rapidly. The radial pulse was regular, 150 per minute.

The heart's dullness extended to the nipple line and there were no murmurs. There were no other signs of heart failure.

Tincture of digitalis was given in doses of 20 minims three times a day and continued for six days, when the patient felt uncomfortable and the pulse was found to have fallen to 86 and was markedly irregular ; the tracings showing that the irregularity was due to a varying number of ventricular systoles responding to the auricular impulses as in Fig. 128. With the cessation of the digitalis the rapid rhythm gradually returned, until four days afterwards the heart had resumed the rapid regular rhythm. Nine days later, that is on July 9, digitalin granules (Nativelle's), one three times a day, were prescribed. After taking thirty-three, he complained of nausea and loss of appetite, and on the 20th the pulse had fallen to 76, and the tracing taken two days later showed that auricular fibrillation had occurred. At this time he was mentally confused for a few days and somewhat delirious. On July 24 the pulse was regular and the heart's action normal, and from this time onwards he gradually regained his strength. Electro-cardiograms taken on May 10, 1910, showed the auricular rate to be 150 and the ventricular rate 300, and those taken on November 16, 1910, showed a normally acting heart.

After that he continued in good health for the three years that he was under observation.

OBSERVATION 27.—Male. Born 1858. Up till the age of 19 the patient was healthy and played games vigorously, but at this age he became disagreeably conscious of intermission of the heart's action. The intermittent action would persist for periods of a month, and was usually worse at breakfast, and also in the night, when he would have to get up, expecting to die. He

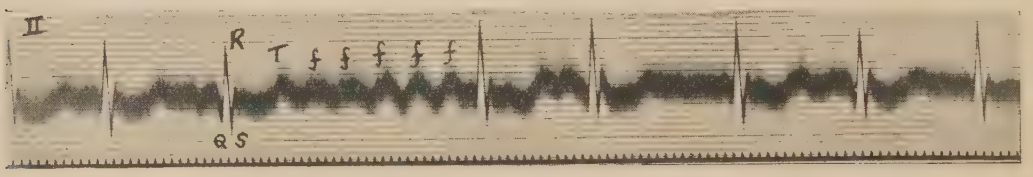


Fig. 140. From the same patient as gave Fig. 139, taken two years later, showing the presence of auricular fibrillation. The auricular fibrillation still continues in 1924.

was very dyspeptic and constipated from this time until he was 32 years of age. He began big-game hunting in 1891, and since then had led a vigorous, strenuous life, and had had a great deal of financial worry and domestic sorrow.

He had never had syphilis, but had malaria in 1891, and an operation for piles in 1911, during which he took gas and ether without trouble.

He had thus been liable to ' heart attacks ' since 1877, which had become somewhat altered of late years. The attacks might come on quite unexpectedly, and without any assignable cause, and might last for an hour or for several days. (The attack in which I found him had lasted ten days.) The rate suddenly increased from 67 per minute to 130-140 ; he was conscious of this acceleration, but it did not cripple him much, and he usually continued his avocation. He said he purposely walked hard for several hours during an attack and actually felt better after it. Various forms of treatment had been tried, but all with no result. I saw him on February 20, 1912. He was a healthy-looking man, but of a spare habit. He complained chiefly of the disagreeable consciousness of the heart's action and a certain degree of lassitude when the heart was so rapid. The pulse was quite regular, 110 per minute. From the electro-cardiograms the ventricular rate was 114 per minute, and the auricular 228. The heart was dull to the nipple line, but no other abnormality could be detected. On February 23 he rested in bed and was given 3 granules of Nativelle's digitalin per day. In thirty-six hours the pulse-rate had fallen to 70 per minute ; then he was given 2 granules per day till the 26th, when the pulse was 68 (10 granules altogether). His doctor wrote to me on March 5 saying the pulse had kept under 76 per minute without digitalis, and the patient had lost all his distress and felt much better. After this the patient led a strenuous life, but enjoyed good health, though he had occasionally attacks of his old trouble, and in 1924 (aged 66) he is still hale and active.

OBSERVATION 28.—Male. Born 1850. His doctor wrote that the patient had enjoyed fair health until the middle of 1910, when he noticed he was gradually becoming short of breath. In January 1912 he had a severe attack of influenza, followed a few weeks later by an attack of gout and bronchitis. When he recovered from these attacks he was very easily exhausted and readily rendered breathless. An increase in the rate of the heart had persisted since his illness, and he was confident it was not present before. The patient had had much business worry during the last few years.

I saw him on March 19, 1912. He was a big stout man with a florid complexion. He complained chiefly of breathlessness and exhaustion on slight exertion. His pulse was small, regular, and 130 beats per minute. There was marked pulsation in the jugular veins, with small waves double the rate of the pulse. The heart's dullness was increased, extending 1 inch beyond the nipple line. The systolic blood-pressure was 160 mm. Hg. An electro-cardiogram showed that the auricle was beating at double the rate of the ventricle. As the condition seemed a recent one, I recommended that he should have a period of rest. This was done, and he again consulted me on May 23, 1912. He stated that the breathlessness and exhaustion were still easily produced, and, in addition, he had attacks of pain striking into the chest on exertion, on one occasion with such violence that he fell on his knees ; the pain lasted only for a few seconds. On four occasions he lost consciousness ; he dropped suddenly down in the street and at once recovered himself ; at another time when at breakfast he suddenly lost consciousness for ten minutes. An examination revealed the same condition as before, that is, a persistent tachycardia of 130 beats per minute, and the peculiar venous pulse, and enlargement of the heart. He was put to bed and given digitalis. The auricular rate was fairly constant, about 270 per minute, while the ventricle was always beating at half this rate. The tincture of digitalis was started on May 24, and continued until the 31st. No effect was perceived until the 30th, when the pulse-rate varied from 114 to 137 per minute, while the auricular rate was 274. This was due to the fact that the response of the ventricle to the auricle varied, sometimes there being one ventricular response to every two auricular beats, or sometimes there being only one to every four auricular beats. On May 31, seven days after starting the digitalis, and after the patient had taken altogether 4 drachms 55 minims, vomiting set in and the pulse-rate fell to 94 per minute, and was very irregular, while the auricular rate continued at 278. The irregularity was due to the varying responses of the ventricle to the auricular beats. The digitalis was stopped for a few days, the pulse remaining irregular. While the rapid auricular rate (260 to 268 per minute) persisted the patient was put on tincture of strophanthus, on June 4. This treatment was continued till June 20, and the patient had taken 15 drachms 15 minims of the drug. It never affected the auricular rate, though the ventricular rate was markedly affected, being irregular and varying at times from 80 to 130 beats per minute, due to the varying response of the ventricle to the auricular beats. After the stopping of the drug the rhythm of the ventricle became regular, about 130 beats per minute, while the auricle continued to beat at double the rate of the ventricle. The flutter persisted, and he lived a quiet life. In 1924 I heard that he had just recovered from an attack of pneumonia (age 74).

OBSERVATION 29.—Male. Aged 61. In July 1912 he had an attack of influenza and pneumonia, and the heart was found to be rapid and irregular. Early in December his doctor found him complaining of a sensation of constriction in the præcordial region. The pulse was 144, with a disorderly rhythm. He was put to bed, and in a few days œdema of the legs and of the base of both lungs set in. Some days later the heart quietened down under digitalis. The heart had been better since, though he had had occasional attacks of tachycardia. In one attack, January 13, 1913, he felt very faint and almost lost consciousness.

I saw him on February 1, 1913, and found him an active, healthy-looking man. The pulse was regular, 80 beats per minute ; the blood-pressure was 175 mm. Hg. The heart's dullness extended ½ inch beyond the nipple line and the sounds were clear.

I saw him again two days later. He said he felt quite well and was not conscious of anything amiss with the heart. The pulse was beating rapidly, 155 beats per minute, quite regularly for short periods, with the pulsus alternans (Fig. 141), then very irregularly. I took a long

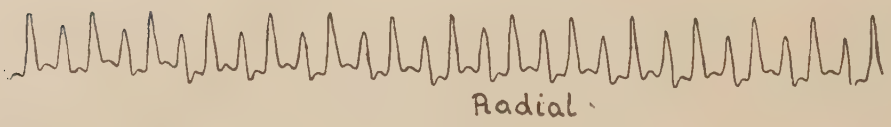


Fig. 141. Pulsus alternans during an attack of auricular flutter. The electro-cardiogram from lead II. in Fig. 143 was taken while the attack was on.

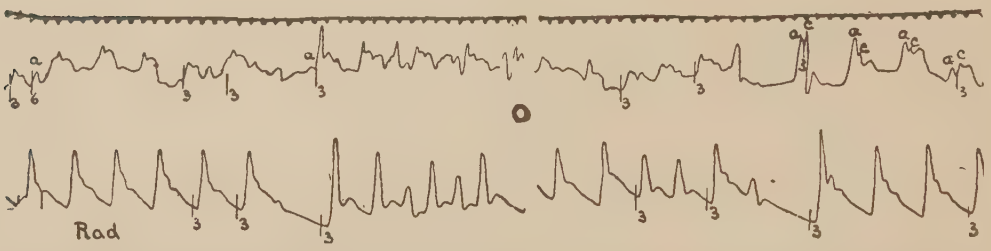


Fig. 142. Tracing of the radial and the pulsation in the neck, showing the beginning and end of a short attack of paroxysmal tachycardia, due to auricular fibrillation. The first six beats and the last three in the radial are regular and due to a normal contraction of the heart. The beats between occur during a period of auricular fibrillation. At *O* a portion of the tracing (representing a period of fifteen seconds) has been cut out. The attack lasted twenty seconds. These attacks varied with periods of auricular flutter (Figs. 141 and 143).

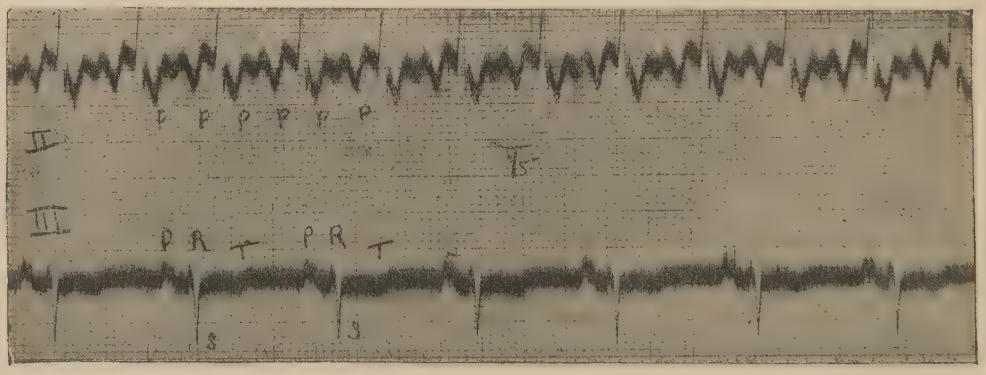


Fig. 143. When the upper record (lead II.) was taken, auricular flutter was present—the auricle (*P*) contracting at the rate of 300 per minute, and the ventricle at half this rate (see Fig. 141). A few minutes later the lower record was taken (lead III.), and the heart had now resumed its normal action—auricle and ventricle beating at the rate of 96.

tracing, and found remarkable variation in the pulse, sometimes rapid and irregular (auricular fibrillation, Fig. 142), and sometimes rapid and regular (auricular flutter), and then for a short time the rhythm would be quite normal (Figs. 142 and 143). An electro-cardiogram taken during the rapid regular period shows the auricle beating at a rate of 308 and the ventricle at 154 per minute (Fig. 143), while the first two leads were taken, but a few minutes later, when the third lead was taken, the heart had reverted to the normal rhythm, one auricular beat to one ventricular beat at the rate of 90 beats per minute (Fig. 143). A later electro-cardiogram taken during the irregular period showed auricular fibrillation. I saw this patient several times; the heart was usually normal in its action, though he was conscious of his heart beating irregularly for several hours at a time. I saw him in one such attack and found the auricle was fibrillating. In 1917 the auricle was fibrillating, and he was very breathless. He had no more attacks of tachycardia, the fibrillation of the auricle persisted, the liver was enlarged, and he was very breathless. He became weaker, and died in 1919.

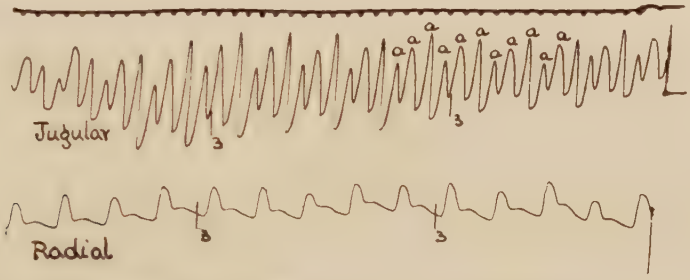


FIG. 144. The auricular rate is three times that of the ventricular = *a*, 270; *v*, 90. (Observation 30.)

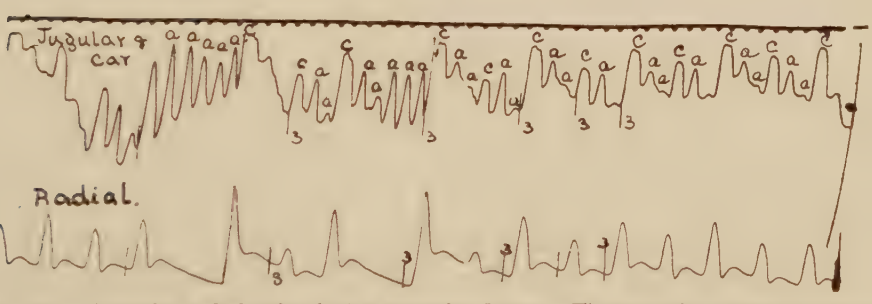


FIG. 145. Irregular radial pulse during auricular flutter. The auricular waves (*a*) are only perceptible during the long ventricular pauses.

OBSERVATION 30.—Male. Aged 62. Consulted me on October 22, 1912. Complained of breathlessness on exertion and swelling of the ankles at night. The breathlessness was noticed first when playing golf in August. He said that he had giddy attacks during the last two years; one bad attack lasted for a minute, when everything went round. His doctor said he had had a large and irregular heart for ten years. He was a big pale man. The pulse was rapid, 90 per minute, and occasionally irregular (Figs. 144 and 145). There was a large diffuse apex beat outside the nipple line. The tracings of the radial pulse (Fig. 146) showed alternation following the extra-systoles, and it is to be noted that the apex beat is just as large and full with a small radial beat as with a large one, in contrast with the apex beat due to an extra-systole (Fig. 146). The jugular tracing (Fig. 144) shows three waves to one pulse-beat, so that the auricle was beating three times as fast as the ventricle. This agrees with the electro-cardiogram. When there was a pause in the ventricle the rapid auricular beats come out very

clearly, as shown in Fig. 145. Digitalis reduced the ventricular rate under 30 (Fig. 147), but did not restore the normal rhythm.

He again consulted me in March 1913. He had been having a long, restful holiday, and was feeling better and fit for work. The heart's action was quite normal, one auricular beat preceding each ventricular beat, the pulse-rate being 75 per minute. Otherwise the heart was in much the same condition as before.

I did not see him again, and he died in 1920 (aged 68). In the last years of his life he became gradually more feeble and short of breath, and was found dead in bed one morning.

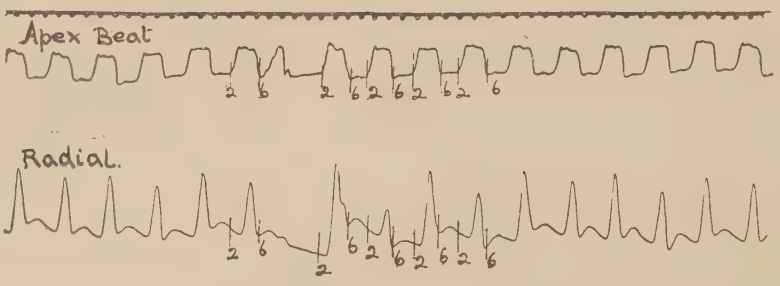


FIG. 146. Simultaneous tracings of the apex and radial in a case of auricular flutter. After an extra-systole alternating character of the radial pulse becomes more marked. Notwithstanding the marked difference in size of the radial beats due to the alternation, there is no difference in the size or duration of the apex beats—in marked contrast to the apex beat caused by the extra-systole.

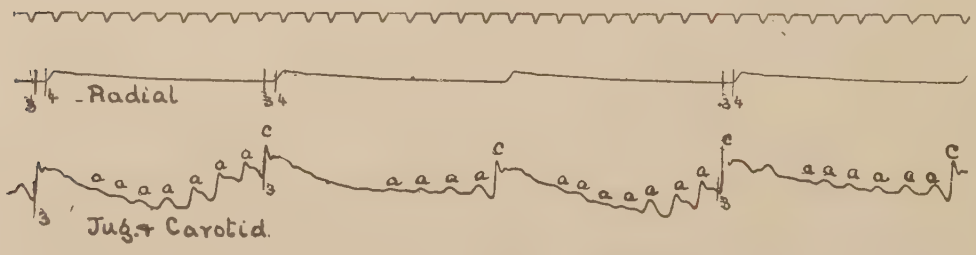


FIG. 147. The radial tracing shows a ventricular rate of 28, and the jugular tracing (*a*) an auricular rate of 280 per minute.

OBSERVATION 31.—Male. Born 1855. He led an active and strenuous life (he was a physician) till the end of December 1909. At this time he had an attack of influenza, during which he worked at high pressure, but seemed none the worse for it and continued his strenuous life. In January 1910 while shaving he noticed that he was breathless and that his cheeks were very blue. He lay down and felt that his heart was beating very rapidly, about 180 per minute. After resting he got up and resumed his work, but in a few days he became so weak and breathless that he had to stay in bed. A few days later he felt better, though the heart-rate persisted at over 140. A tracing taken on January 21, 1910, showed a pulse-rate of 170. At the end of January he went away for ten days' rest, and returned on February 6, and though he resumed work he was weak and breathless. I saw him on February 13. He was able to be up, but was very breathless on going upstairs. The pulse was very irregular, about 75 per minute. The tracing from the neck showed numerous small waves, characteristic of auricular flutter. The heart's dullness extended to the left nipple line, and the sounds were free from murmurs. Save for the breathlessness on exertion, there was no sign of heart failure. Two days later an electro-

cardiogram was taken which showed a regular ventricular rhythm with a rate of 145 per minute, the auricular rate being 290 per minute—exactly double.

After this date the patient continued at work, and on February 18 he had a very busy and worrying day, and had to run to catch a train. On the 19th he felt ill and his temperature was 100° F. On the 20th the temperature was normal, and the pulse was small and very irregular and many beats imperceptible. The heart-beats were 180 per minute and very irregular. There was marked pulsation in the epigastrium; the heart's dullness extended ¾ inch outside the nipple line; the veins of the neck were full and engorged. During the night the breathing was difficult and Cheyne-Stokes in character. In the morning of the 22nd the pulse-rate was 180, and in the evening it fell to 104. For the next few days the pulse remained very rapid, and the condition of the patient became worse, with orthopnœa, Cheyne-Stokes respiration, and at times partial loss of consciousness. On February 26 the heart suddenly reverted to the normal rhythm, falling to between 60 and 70 beats per minute, and all the symptoms of heart failure speedily passed off. After this he felt very well, and kept so until he had a return of the tachycardia on March 8. This continued for a few days, then suddenly disappeared. From this date he gradually improved and went for a month's holiday. He returned to work and kept fairly well till the summer of 1912, when he again became conscious that the heart was acting abnormally, but he still pursued his strenuous life and went for a holiday in August, but had to return home and go to bed. When I saw him in September the rate of the heart was rapid, about 117, and irregular and characteristic waves of auricular flutter were found in the jugular tracing. He was slightly livid and lay propped up in bed. At times he had Cheyne-Stokes respiration, and the heart dullness extended to 2 inches outside the nipple line.

He had been taking digitalis and the dose was increased, with the result that the ventricle became slower, and he gained some strength and made a partial recovery, but the auricular flutter persisted; the heart always tended to increase in rate, but was kept in check by the digitalis, till his death occurred suddenly in January 1913.

OBSERVATION 32.—Male. Born 1833. He enjoyed good health until 1901, when he began to have attacks of loss of consciousness. In one of these attacks he fell, and was found to have dislocated his shoulder. As a rule the attacks came on quite suddenly; he would fall and immediately recover, but would feel dazed and weak and be conscious of a feeble action of the heart. As a rule the attacks readily passed off. In 1906 he partook of a hasty lunch, and then hurried half a mile to catch a train. After getting into the train he became unconscious, and at the next station he was carried to an hotel, where he lay unconscious for several hours and with a pulse so small that the doctor in attendance thought on several occasions he was dead. He recovered, and had a few much slighter attacks since. He was conscious at times of his heart running away, but he kept quiet and it passed off after a few minutes or a few hours.

I saw him on February 21, 1911. He was a tall, spare man, with a blue nose and cold fingers. The pulse was occasionally interrupted by extra-systoles. The heart's dullness was not increased to the right, but extended to the nipple line on the left. The apex beat was somewhat diffuse and forcible. There was no murmur, but the first sound at the apex was soft, and when he lay down it was inaudible.

I saw him again on August 2, 1912. He was suffering from great prostration, due to an attack which had come on thirteen days previously and had continued till I saw him. He was very pale and distressed-looking, and lay propped up in bed; the pulse was barely perceptible and so rapid as to be uncountable. The deep jugular veins were full and pulsated largely. From the tracing the rate was just over 200 per minute, and I had the greatest difficulty in getting a record of the radial pulse, while a record was easily obtained from the jugular. The heart's dullness was at this time outside the nipple line, there were no murmurs; the first sound was scarcely audible and the second sound was markedly accentuated. There was no dropsy or enlargement of the liver. The patient was put on digitalin granules, one being given every six hours. This was continued for three days, when the pulse-rate fell to 100 per minute. The patient became very restless and his mind wandered. The digitalin was stopped. After this various remedies were tried, but none seemed to have a definite effect. The pulse-rate for the

next month varied markedly in rate, being 180 beats per minute, and sometimes falling as low as 74; sometimes quite regular, at other times irregular. As I did not see him after August 2, I am not able to say whether the abnormal action was continuous all that time, but from the account of the rate and rhythm given by his doctor I should think it was fairly persistent, it may be with intervals of the normal rhythm. The patient's general condition varied much; he was at times fairly comfortable, but often suffered from severe prostration. Slight excitement or exertion would often induce an attack of very rapid heart action. He gradually became weaker and died.

OBSERVATION 33.—Female. Aged 39. Admitted to hospital on September 22, 1912, complaining of shortness of breath, pain in the abdomen, and swelling of the legs. This had been present for three and a half months, when she first noticed shortness of breath, and during the last six weeks her legs had swollen. There was no history of rheumatic fever, but a Wassermann reaction was positive.

On admission there were slight dyspnœa, œdema of the legs, and cyanosis of the lips. The apex beat was in the sixth space, $6\frac{1}{4}$ inches from the mid-line. The limits of the heart's dullness were 1 and $6\frac{1}{4}$ inches to the right and left of the mid-line, in the fourth space. There were systolic and diastolic aortic murmurs audible over all the præcordium. There were crepitations

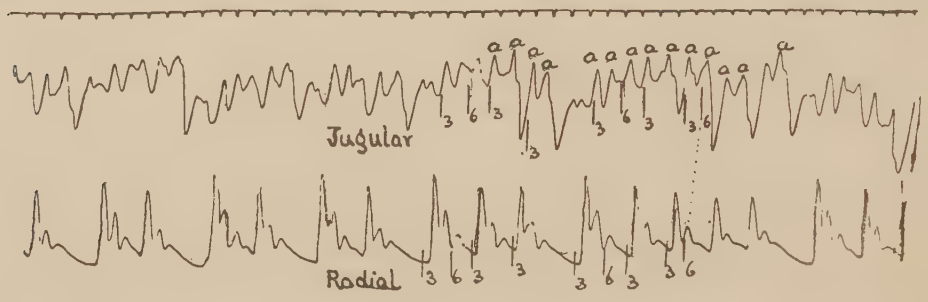


FIG. 148. Shows an irregular radial pulse, and a jugular pulse with numerous auricular waves.

at the base of both lungs. The pulse was regular, 90 per minute. The liver extended 1 inch below the costal margin in the right mid-clavicular line. The urine was acid, 1020, and albumin was present.

On rest in bed she showed improvement which lasted for seven days. Then she had attacks of dyspnœa, associated with perspiration and with pain over the præcordium. The liver increased in size, and there was tenderness over it. She was placed on tincture of digitalis on October 16, 1912, 1 drachm per day; on the 23rd the digitalis was discontinued because of nausea and vomiting. There was slight improvement in the general condition and a decrease in the amount of œdema, but no slowing of pulse-rate or increase of blood-pressure.

On November 18, 1912, the administration of digitalis was again commenced; on the 25th, during its administration, the character of the jugular pulse became altered, and showed the presence of auricular flutter. Her general condition gradually became worse, there was more œdema and ascites. She left the hospital on December 4, and died ten days later at home. The auricles were in a state of flutter when she left the hospital. After the onset of auricular flutter the general symptoms became worse, but no change was found in the area of cardiac dullness, or the character of the murmurs, but the pulse became irregular (Fig. 148).

OBSERVATION 34.—Male. Aged 63. Consulted me first on April 27, 1910. The patient felt quite well until November 1909, when he was troubled with a cough and shortness of breath. In the last few months he had been troubled with attacks of great breathlessness occurring about 3 A.M. These attacks were very severe and lasted for about half an hour. The attacks were accompanied by coughing and some phlegm. The patient was a big, powerful-looking man.

The pulse was large and the arterial wall thickened ; the blood-pressure was 125 mm. Hg. There was an occasional extra-systole, followed by the pulsus alternans for a few beats. The heart was enlarged and the apex beat was 2 inches outside the nipple line. The first sound was reduplicated. A tracing of the jugular pulse shows an increased *a-c* interval (Fig. 149), and electrocardiograms showed an increase in the *P-R* interval, and the changes characteristic of damage to the right branch of the bundle, that is, the left ventricle started contracting before the right.

The patient denied a history of syphilis, but certain scars on the leg had a suspicious appearance. There was a slight trace of albumin in the urine. The patient improved under treatment and kept fairly well until July 1912, when he had an attack of gout which weakened him. I had seen him from time to time during the interval and detected little change. I saw him on April 5, 1912, and he felt weak, but could play a game of golf with comfort. In January 1913 he went abroad and was taken seriously ill, with great weakness and breathlessness. I saw him on March 2. He lay propped up in bed in a slight stupor, with marked Cheyne-Stokes respiration, and the pulse was very rapid, varying in rhythm, periods of regularity varying with periods of irregularity. Slight waves in the jugular pulse, indicative of auricular flutter, were present at this time.

The patient's condition did not improve, and he gradually sank and died in May 1913.

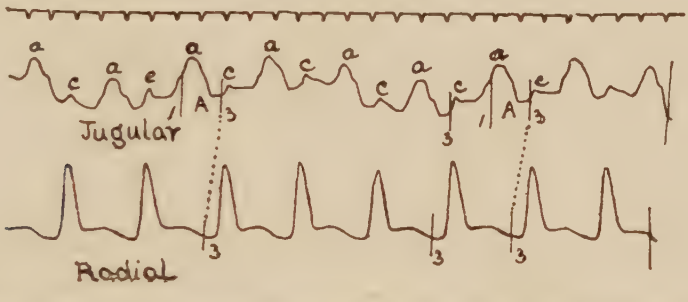


FIG. 149. Simultaneous tracings of the jugular and radial pulses, showing a wide *a-c* interval (space *A*).

OBSERVATION 35.—Male. Aged 47. Seen on December 4, 1912. The patient had scarlet fever in childhood, which left him slightly deaf. He had had good health and led a temperate life. He was quite well up to November 28, 1912, and on that day he played a game of golf and enjoyed it. That evening there was a slight rise in his temperature, and he coughed up some blood-stained sputum. The next day he was rather breathless and dusky. On December 2 he had an attack of great breathlessness and the pulse became rapid, irregular, and at times scarcely perceptible. From the breathlessness he recovered in an hour. When I saw him on the 4th he lay propped up in bed, the face of a dusky tinge, the breathing slightly laboured and becoming markedly so in turning over. The heart's dullness extended to the nipple line, the action was irregular and the sounds muffled. Above each clavicle the jugular bulb was seen beating so forcibly as to be perceptible to the touch, and giving rise to a short sharp sound. A tracing showed the jugular pulse to be occasionally irregular, and each large wave in the jugular occurred immediately before a radial pulse-beat (Fig. 150). There was impaired resonance of the bases of both lungs and a few fine crepitations.

He improved in his general condition from this date, though his pulse was very irregular at times. In a few days albumin appeared in the urine, and the quantity of urine passed gradually diminished. Still he seemed to be mending, and on January 16 he was able to get up, but was very short of breath, and the heart's action continued very irregular.

From this date he gradually became worse, Cheyne-Stokes respiration developed, the urine became scanty and dropsy appeared, extending gradually up the legs to the abdomen. The

heart's dullness increased in size and a mitral murmur developed, and the rhythm continued irregular. The Cheyne-Stokes respiration was very marked, and during the apnœic stage I obtained tracings from the jugular pulse, showing two well-marked waves to one ventricular beat (Fig. 134). He gradually sank and died on April 2, 1913.

OBSERVATION 36.—Male. Aged 33. On one occasion in 1912 he fainted while running. Apart from this isolated syncopal attack, he was well until about October 1913, when he began to notice shortness of breath at his ordinary heavy work of stoking. Palpitation and dizziness were subsidiary symptoms. He had no pain and no swelling of feet. Five months later, and after absence from work for a month, he was admitted to hospital. No history of acute

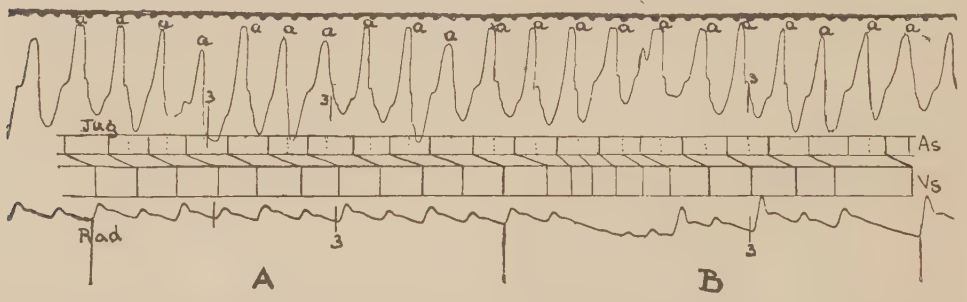


FIG. 150. Tracing of jugular and radial pulsations during auricular flutter. The intercalated diagram suggests the explanation, that in addition to the large wave, *a*, in the jugular, there was another auricular wave which occurred during the ventricular diastole (see Fig. 151). The irregularity in the radial tracing is probably due to the ventricle responding to each auricular beat for a short period. The periods *A* and *B*, though of the same duration, contain the same number of auricular beats but include a different number of ventricular systoles, a characteristic of auricular flutter.

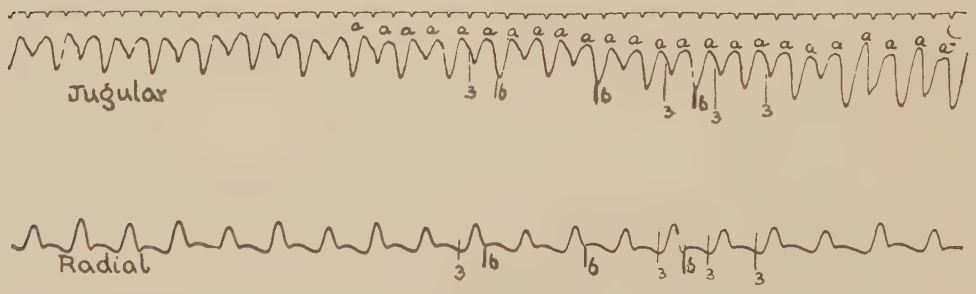


FIG. 151. The auricular rate (*a*) is about 240, and the ventricular one-half of this.

rheumatism or chorea was obtained, and the Wassermann reaction, in February 1914, was negative.

February 16, 1914. He looked fairly well, and was not breathless except on exertion. The apex-beat was in the fifth space, just external to the nipple line. At the apex the first sound was accentuated; at the apex and in the pulmonary area the second sound was duplicated; no murmurs were heard on this date. No abnormal signs were discovered in the lungs or abdomen. Œdema was absent, the urine was free from albumin, and the systolic blood-pressure was 110 mm. Hg. The pulse was 68, and regular. The jugular tracing appeared to be normal, with 'a.c.v.' in sequence, yet an electro-cardiogram demonstrated auricular flutter, A : V : 4 : 1. Rest in bed without drugs for one week had no influence on the flutter or the pulse. A diastolic murmur was now heard at the apex (mitral stenosis).

For the next eighteen days tincture of digitalis, 1 drachm daily; then stopped on account of vomiting; long pauses in the radial pulse (increased block) (Fig. 132). Two days after digitalis had been stopped *auricular fibrillation* appeared, and it persisted thereafter (Fig. 152).

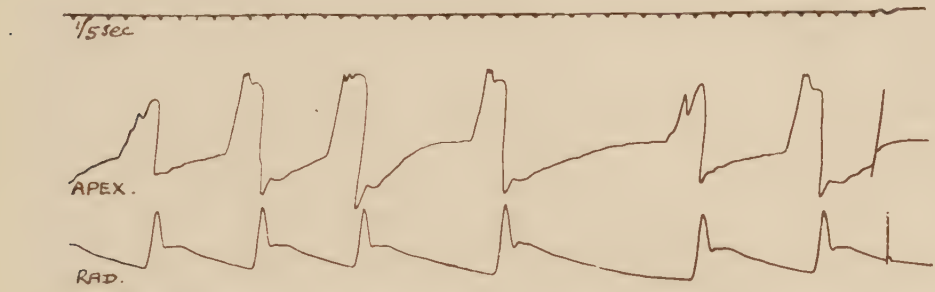


FIG. 152. From the same patient as gave Fig. 132. Two days after the digitalis was stopped the heart passed into fibrillation; with the persistence of the same kind of ventricular irregularity the flutter waves have disappeared.

For two months longer he was detained in hospital by three separate attacks of pulmonary infarction. A month later fibrillation was still present. He was unfit for work. After the onset of war no further observations were made. Four years later, in 1918, he died at home of 'heart failure after influenza'.

CHAPTER XXXVII

The Origin and Nature of Auricular Flutter

The origin and nature of auricular flutter.
The ' circus rhythm '.
The nature of the contraction in auricular flutter.
The impulses discharged by the auricle.
Origin of the paroxysmal attacks.

As has already been pointed out, the view taken in this book is that auricular flutter arises in the same way as auricular fibrillation, being due, not to any disease of the auricle, but to loss of control of the sino-auricular node and the parts of the genetic system in the auricle.

With the cessation of the activity of the s.-a. node there follow all those other consequences which were described in auricular fibrillation—*i.e.* an absence of control over the a.-v. node—with the result that it becomes much more susceptible to all agents that increase or decrease its activity. On account of the peculiar character of this node its effect upon the ventricle is to stimulate it in such a way that its contraction tends to become ineffective and imperfect, and produces the same form of heart failure as occurs in auricular fibrillation. The a.-v. node in this condition offers an extraordinary opportunity for investigating vital activities, and it has been already used for this purpose in different sections of the book. But there is still a great deal more information to be derived from this study, of which only a small part can be dealt with here.

The nature of the auricular contraction.—There is something peculiar about the contraction of the auricle in auricular flutter. The force of the beat is, as a rule, very slight, although on rare occasions large waves can be detected extending up the jugular veins (Fig. 127), while they can make an impression upon the apex-beat (Fig. 132). So far I have never found them give rise to a murmur in mitral stenosis (see Observation 36, p. 232).

There is also some difficulty in understanding how the rhythmic waves are brought about. It is quite possible that they correspond to a similar phenomenon which has been described by Langley and Walker as occurring in fibrillation of the voluntary muscles. Langley states that in fibrillation movements may be so fine that they are only revealed by a magnifying glass, but at times they become much larger and occur in a series of regular movements of a considerable size. As there is a tendency for flutter to pass into

fibrillation, and even in some cases for short periods of the one movement to intermit with short periods of the other, there are some grounds for such a suggestion.

The 'circus rhythm'.—Lewis, by an ingenious use of the electro-cardiograph, has obtained records from which he concludes that in auricular flutter of the human heart the contraction of the auricle is due to the presence of a contraction, or excitation wave, which passes continuously round the wall of the heart in a circle. This circus movement is evidently of the same kind as that which occurs in the jelly-fish already described (p. 63), when a ring of the body wall is separated from its connections with the marginal sensory bodies. One does not quite see how this occurs in the human auricle; the wall of the jelly-fish consists mainly of a network of nerve fibres, overlaid by a layer of muscle fibres—the impulse travelling round by the nerve network. Such an arrangement has not been found in the auricle, although there may be some modification of the genetic system which leads to the same result.

If the view is established that there is this 'circus' rhythm, there is wanted an explanation of how it comes about. Those who accept it seem to think that the discovery of the fact explains the fact. What is wanted is a knowledge of how it comes about, and what are the structures involved in its production.

The nature of the contraction in auricular flutter.—The view put forward here (pp. 62 and 63) is that flutter and fibrillation of the auricle are identical with the movements in voluntary muscle and in the jelly-fish, when the muscle is separated from the structures which regulate and control the muscle.

The contraction of the auricle in auricular flutter is evidently quite different from the normal.

If the description given by Keith of the peculiar effect produced by the contraction of the tenia terminalis and the pectinate fibres, described on p. 92, be correct—and the evidence that Keith adduces certainly favours his explanation,—there cannot be in auricular flutter that sequential contraction of these and other structures which is necessary for the normal functioning of the auricle.

The impulses discharged by the auricle.—Attention has been called to the reactions which are produced by impulses which arise in organs specially developed for the purpose. Impulses from the cortex of the brain cause a contraction of the muscle, purposive and graded to suit the circumstances of the moment. Impulses which start from the s.-a. node result in a similar contraction of the ventricles. It has also been pointed out that such graded contractions cannot be produced by impulses which originate from any other form of stimulation. Electrical stimulation of nerve or of bundle cannot give the same kind of reaction.

Evidence has already been given that the muscle cells of the auricle discharge impulses in auricular flutter which stimulate the a.-v. node to discharge in its turn, impulses which result in a ventricular contraction.

The question arises, Are the effects produced by these auricular impulses the same as those arising from the sino-auricular node? This question can be answered in the negative if we appreciate what happens in the series of records in Figs. 7, 8, and 9. In Fig. 7 it is seen that the stimulus which excites the a.-v. node proceeds from the auricle, but every beat of the ventricle differs from that which precedes and follows it—producing an alternating rhythm of strong and weak beats—that is, of beats in which more fibres of the ventricle participate at one time and fewer at another.

When the rate falls below 150 the beats begin to vary still more in size, as in Fig. 8. This variation is not due to a variation in the length of the diastolic pause—*i.e.* to variations in the contents of the ventricle—for beats preceded by pauses of the same length vary in size.

These variations are not found in hearts which are under the control of the s.-a. node, so we must conclude that, as all impulses are the same, some other influence is exercised by the s.-a. node—and this is one of the reasons for suggesting an influence which regulates and controls the contraction.

This view was indicated in describing the changes that accompany auricular fibrillation, but in auricular flutter we get a better insight into the matter, because the number of impulses (or it may be more correct to say the number of groups of impulses, as each cell in its contraction throws out an impulse) can be counted. The varying reactions (the contractions of the ventricle) are due to differences between the number of impulses and the excitability of the cells which receive them. In auricular flutter the number of impulses is constant, so that the variations in the ventricular contraction are due to differences in the excitability of the a.-v. node.

We see, therefore, that the varying sizes of the pulse-wave in the radial tracings in Figs. 6 and 8 are due to variations in the excitability of the a.-v. node. As these variations in the size of the pulse beats indicate variations in the number of muscle cells participating in the contraction, we thus are able to trace the source of the varying contractions of the ventricle to the a.-v. node.

The significance of this observation is that the same process is at work in producing the slowing in response to rest as to digitalis. It was reasoned that the slowing on rest was produced by increased activity of the vagus with diminished activity of the sympathetic, and it is reasonable to conclude that digitalis exerts its influence through the vagus nerves.

Another matter which also comes out of this consideration is the extraordinary susceptibility to digitalis of the a.-v. node when the control of the s.-a. node is lost. It is in auricular flutter that I have found the greatest slowing in response to digitalis. In Fig. 147 the pulse-rate is under 30 while the auricular rate remains at 300—a result, so far as I know, never obtained in a heart while the s.-a. node controls the rhythm.

Origin of the paroxysmal attacks.—In most cases auricular flutter is intermittent in its onset before it becomes permanent. This period of intermittent attacks may go on for many years, as shown in Observations 22 to 24.

In other instances it may precede the permanent establishment of auricular fibrillation, when the paroxysms cease. The fact that the condition is permanent and may last for many years indicates that organic changes have taken place which have destroyed the functional efficiency of the s.-a. node. The temporary attacks would therefore indicate the presence of a slight degree of impairment, while the progress of structural changes is indicated by their more frequent occurrence till flutter or fibrillation is permanently established.

CHAPTER XXXVIII

Heart Failure in Auricular Flutter and Prognosis

Syncopal attacks.
Respiratory symptoms.
Prognosis.

The symptoms of heart failure in auricular flutter are the result of a diminished output of blood from the heart, and are to be found in the organs whose functional efficiency is impaired. The most striking of these symptoms are those due to the nervous and respiratory systems.

Syncopal attacks.—The sudden onset is sometimes accompanied by such a defective output that the patients lose consciousness, as described in Observations 22, 23, and 32. The deficient output which produces these attacks can be seen from certain pulse records. Partial loss of consciousness may occur in individuals where the condition has been established for some time. Fig. 134 is taken from a man (Observation 23) who had frequent

Fig. 153. Was taken from a patient during an attack of auricular flutter. The pulse disappeared after a brief period, and the patient partially lost consciousness. During the time the pulse disappeared there are indications in the tracing of movements that the pulse-rate had increased to nearly 300 beats per minute.

attacks, for many years, of auricular flutter, during which he became unconscious. Fig. 153 is part of a tracing taken by Dr. Orr. The patient seemed to be quite well, when suddenly for a brief period he felt as if he were about to faint. This coincided with the disappearance of the pulse from the wrist, shown in the tracing. This tendency for the pulse to disappear is not an infrequent event, although it rarely proceeds so far as that the patient loses consciousness.

The reason for this great weakness of the pulse is almost certainly due to the ventricle responding for a brief period to every auricular contraction. One can infer this from the consideration of Figs. 134 and 153, where there are

slight indications of the pulse-wave occurring at regular intervals. I had the opportunity of seeing a patient, described in Observation 4, on several occasions when the pulse attained a speed nearly of 300 beats per minute. The overpowering sense of weakness was the chief sensation felt by the patient, accompanied by the feeling as if he were going to lose consciousness.

Respiratory symptoms.—Breathlessness brought on by effort, and even when lying in bed, is another of the most common features of this complaint. The slightest effort will sometimes produce such distress that it may be taken for an attack of asthma. One day a doctor called to see me, suffering from this condition, and while I was examining him a colleague, who was an authority on diseases of the lung, came into the room. My patient had just been laid on the couch, and the effort brought on such distress that my colleague at once diagnosed the case as one of asthma, and began to give me instructions with regard to the treatment.

From the histories that have been given it will be seen that the degree of breathlessness varies very considerably. Quite a number of patients suffer very little, and then only when a considerable effort is made. The reason for this is not always quite clear, but one can understand it when the pulse-beats are small and imperfect, the contraction of the ventricles being partial and incomplete.

In many of these cases the lung is the first organ to show the physical signs of heart failure, beginning with slight œdema, and sometimes rapidly progressing till there are dullness at the bases and expectoration of blood. I have seen such cases diagnosed as pneumonia by experienced consultant physicians. The recovery of such patients, when the heart resumes its normal rhythm, is quite dramatic.

Here, as in auricular fibrillation, there occur those symptoms of heart failure which speedily supervene on the onset of flutter, described on p. 223.

The cause of this heart failure is due to the extreme weakness of the ventricular contractions, as in auricular fibrillation.

Prognosis.—From the description given of the illustrative cases in Chapter XXXVI. it will be seen that the prognosis is as varied as the symptomatology. We can conclude that, though auricular flutter may embarrass the heart in its function, it is not in itself a dangerous condition. The danger here, as in auricular fibrillation, lies in the heart being already the seat of extensive disease especially affecting the ventricles, or valvular disease of a kind that adds to the embarrassment of the ventricles. The weakened ventricle, burdened with the abnormal rhythm, is unable to cope with its work. There is also an element of danger in the number of ineffective contractions of the ventricle.

In giving a prognosis, therefore, each case must be carefully studied by itself, and the condition of the ventricle and the valve defects recognized, and particularly the manner in which the efficiency of the circulation is maintained.

The neglect of these principles may result in the doctor giving a wrong

prognosis. About a dozen years ago I was called to see an elderly man who had evidently suffered from an attack of auricular flutter during the night preceding my visit. At that time I had seen a large number of people at different ages with these attacks, and had come to the conclusion that in themselves they were without danger, and so I gave a favourable prognosis. The following night the patient had another attack, in which he died. I was surprised, and reviewed in my mind all the features of the case to see where I had gone wrong. I had little difficulty in finding the source of my error ; I had neglected to give due weight to the patient's response to effort before the attack, and to the conditions of the circulation while the attack was on. Although a seemingly healthy man, his response to effort was greatly limited because of breathlessness and pain. During the attack he coughed up blood-stained sputum. These were evidences of such cardiac impairment that the superadded burden of the abnormal rhythm was more than the heart could successfully overcome.

Since that time I have had a considerable number of patients with angina pectoris who have developed auricular flutter, and it has even a more disastrous effect than auricular fibrillation, every one of them drifting and dying within a few months of its onset—the reason being that the ventricular muscle was greatly damaged.

I have seen a few patients with obscure febrile affections develop auricular flutter which has terminated fatally. In them probably the agents of disease have also attacked and impaired the heart muscle as well as the s.-a. node. When there is much distress present and when the flutter is persistent, prognosis should be deferred until treatment is tried (see Observation 25).

Grave symptoms may appear while the patient is liable to paroxysmal attacks. When the condition passes into fibrillation the attacks cease and the prognosis is then that of auricular fibrillation (see Observations 24 and 36).

CHAPTER XXXIX

Treatment of Auricular Flutter

Digitalis.
Danger of digitalis.
The fibrillation that follows auricular flutter.
Observations.

I know nothing that will prevent or cut short the paroxysmal attacks of auricular flutter when the attacks are of short duration. I have tried many remedies; and occasionally I have found one thing which seems to shorten or stop the attack in one patient but is without effect in others. There can be little doubt that some patients do contrive means of stopping the attacks, some by one method and some by other methods. One patient, for instance,

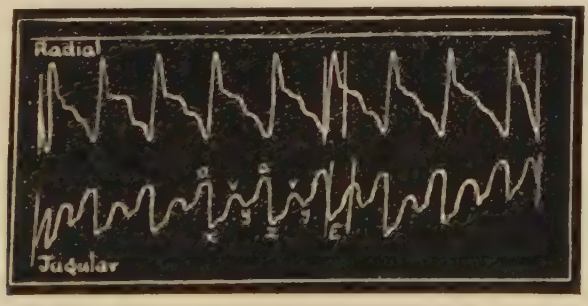


Fig. 154. Shows a normal rhythm at a moderate rate. (Observation 37.)

would stop an attack by bending down and taking a deep breath; another by inducing vomiting. This patient's experience was so instructive that it is quoted at some length at the end of this chapter.

Digitalis.—When the attacks last for a long time and seem to be permanent, we can sometimes bring them to an end by the use of digitalis. As this effect of digitalis is so remarkable and reveals so much that is of value in regard to certain vital processes, to the functions of the a.-v. node, and to the action of digitalis, a somewhat full description is necessary.

My attention was first drawn to this effect of digitalis twenty years ago.

OBSERVATION 37.—In March 1903 I was attending a girl, aged 16, who suffered from breathlessness and palpitation on exertion, with slight œdema of the legs. There was a history of rheumatic fever. The heart was regular—86 per minute—the venous pulse was of the auricular

type (Fig. 154). The heart was greatly enlarged, and there was present a loud, rough, systolic, mitral murmur. With rest and digitalis she gradually improved. The following year she began to have attacks of heart failure, becoming cyanosed, with enlargement of the liver and great œdema of the legs. The rate became greatly increased, which I attributed to auricular paralysis. When in this condition she became remarkably susceptible to digitalis, the pulse-rate speedily falling on its administration, with great improvement in the condition at the same time. At that time I had not recognized the difference between auricular flutter and auricular fibrillation.

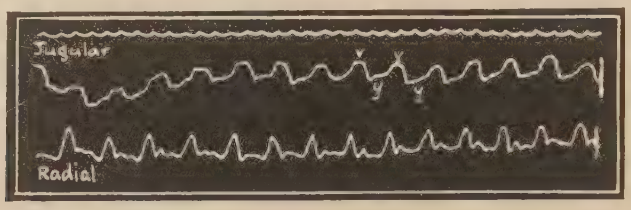


Fig. 155. Showing a rapid but regular pulse, the jugular being of the ventricular type.

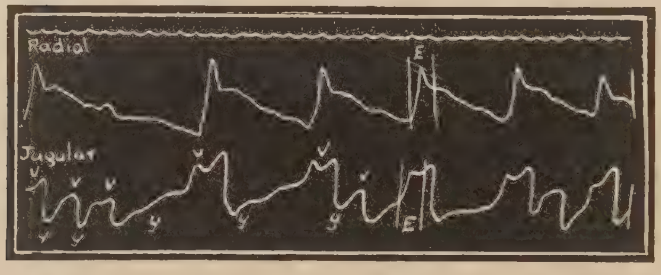


Fig. 156. Shows the effects of digitalis. (Observation 37.)

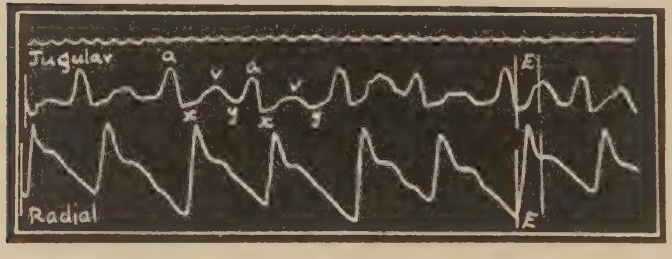


Fig. 157. Shows the return of the heart to the normal rhythm under the influence of digitalis.

The examination of these records now leaves little doubt that these attacks of extreme heart failure were due to the onset of auricular flutter. Thus, in one attack, the pulse became small and rapid, but quite regular (Fig. 155). On April 4, 1904, she was put upon Nativelle's digitalin granules, $\frac{1}{210}$ grain, and given one a day. The drug had no effect till April 14, when the pulse became slow and irregular, the rate being 35, the jugular pulse being double that rate (Fig. 156). The digitalis was stopped, but as the pulse-rate rose to 80 per minute on April 17 the same dose of digitalis was prescribed. The patient's condition had considerably improved, but as the pulse did not respond, the dose was doubled on May 1, and continued till May 14. During this interval I had not taken any tracings. On the 15th I took a tracing, and found that the heart had resumed the normal rhythm (Fig. 157). I then recognized that this patient's

susceptibility to digitalis varied according to whether she had paralysis of the auricle, as I called the condition then, or normal rhythm. During the next year she had a number of attacks of extreme heart failure, and the digitalis always acted in the same way, producing auricular fibrillation and slowing the pulse, but having no effect on the rate when the rhythm was normal, until in the last months of her life the drug lost its effect.

The next case of this kind I observed is described in Observation 24.

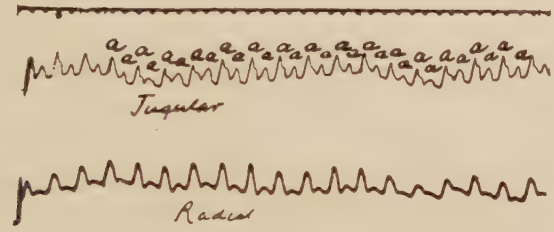


Fig. 158. Tracings of the jugular and radial pulses during auricular flutter. The waves (*a*) are due to the auricle, and are at the rate of 300 per minute. The alternation in the size of these waves is due to one beat of the auricle falling during ventricular systole, giving rise to the larger waves, and the other beat falling during the ventricular diastole. The rate of the radial pulse is 150, exactly half the auricular rate. The reason for this is shown in the electro-cardiogram (Fig. 131). The effect of digitalis upon the patient is shown in the next three records following (Figs. 159, 160, 161).

A similar result is shown in Figs. 158-161. It will be seen in Fig. 158 that at the beginning of the treatment the radial pulse was at the rate of 150 per minute and the auricle 300. The patient was given 1 drachm of the tincture of digitalis per day and it slowed the pulse-rate as in Fig. 159,

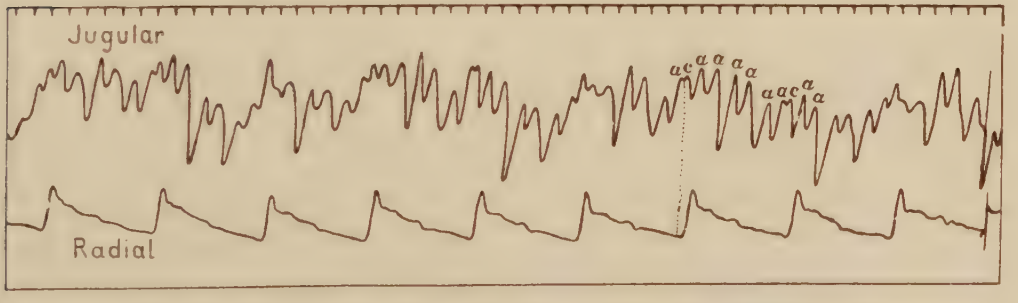


Fig. 159. Showing the effect of digitalis in reducing the ventricular rate while not affecting the auricular.

but it had no further effect for six days, when the auricle passed into fibrillation as in Fig. 160, the digitalis being then discontinued. Five days later the pulse became regular and the rhythm normal as in Fig. 161.

The same results are shown by the electro-cardiographic records in Figs. 162, 163, 164.

There are a number of points, some of which are not clear, as to the manner in which the reaction to digitalis is brought about.

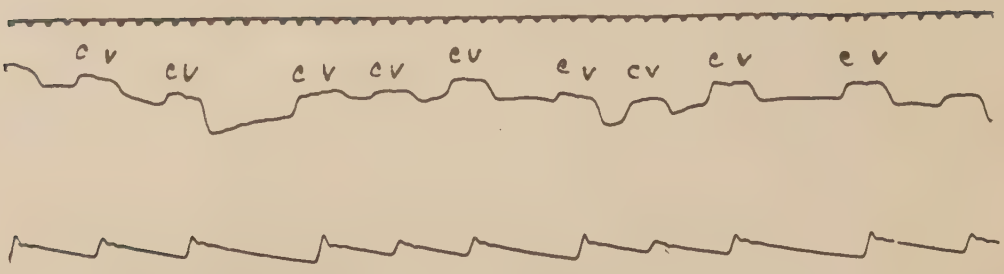


Fig. 160. The further effect of digitalis was to produce auricular fibrillation—the flutter-waves have gone and the venous pulse is now of the ventricular type.

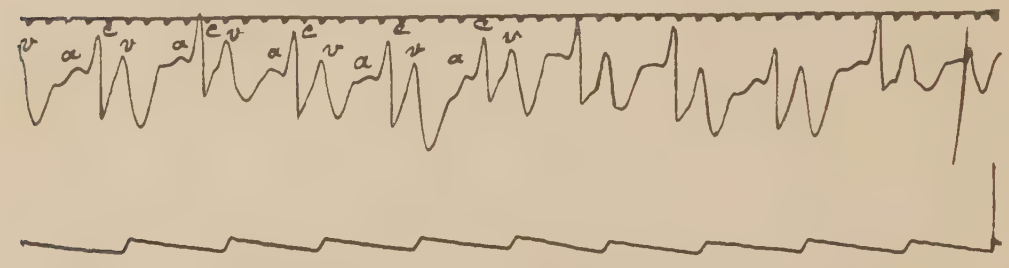
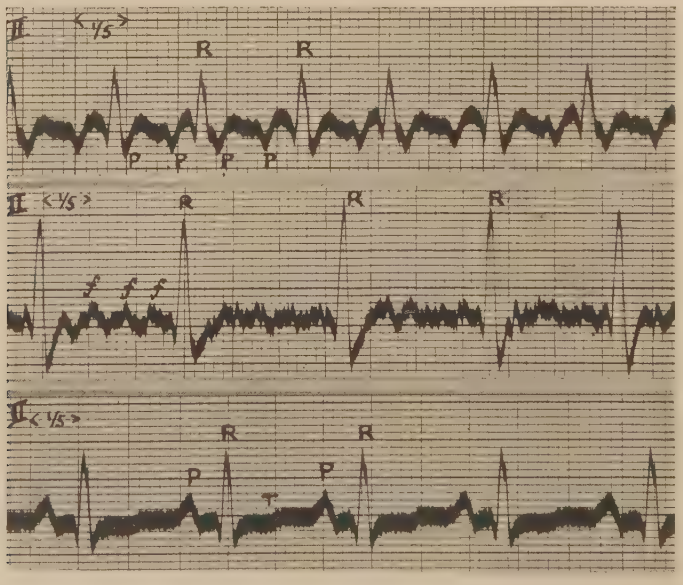


Fig. 161. Five days after stopping the digitalis (after Fig. 160 was taken) the normal rhythm returned.



Electro-cardiogram showing the effects of digitalis in auricular flutter (J. Parkinson).

Fig. 162 shows auricular flutter ; Fig. 163 auricular fibrillation ; Fig. 164 the normal rhythm.

The decrease in the rate of the ventricle when auricular flutter and auricular fibrillation are present is not due to the digitalis producing 'heart-block' in the ordinary sense of the term (see p. 251). It acts through the vagus and diminishes the excitability of the a.-v. node.

In the description given of the manner in which the a.-v. node acts in auricular fibrillation and flutter the node is shown not to be a conducting structure, but one which generates and discharges impulses just like the s.-a. node. Every one recognizes that it would be incorrect and misleading to state that the s.-a. node was blocked when digitalis, or rest, or any other agent decreased the rate of the pulse. It is equally incorrect and misleading to state that digitalis, or rest, or any other agent decreases the rate of the pulse in auricular fibrillation and flutter by inducing block.

In this connexion there is an interesting observation which shows the difference between this effect of digitalis and heart-block. It will be shown later that digitalis sometimes produces what is commonly called partial heart-block, shown by an increase in the length of the *a.-c.* interval. When patients with auricular flutter are given digitalis and the pulse-rate falls to between 40 and 50 beats per minute, and the auricle passes into fibrillation, after which the normal rhythm is suddenly resumed, the rate of the pulse immediately increases as shown in Figs. 138 and 161, *but there is no increase in the a.-c. interval.* In Observation 24, for a day or two when the normal rhythm had been resumed it was continuously interrupted by periods of fibrillation with a rate of about 40 per minute, but the *a.-c.* interval during the periods that normal rhythm was present showed no increase (Fig. 138).

From this observation we draw the conclusion that the influence of the s.-a. node upon the a.-v. node is such that it compels the latter to increased activity even when its excitability has been decreased by the action of digitalis.

Practically in every case that I have been able to follow long enough this restoration to the normal rhythm was not permanent.

Although in many cases the digitalis fails to produce this reaction, it still has a beneficial effect in slowing the rate of the ventricle, and so enabling the ventricle to produce more powerful and effective contractions. The maintenance, for instance, of the pulse at the rate shown in Fig. 159 with such forceful beats would be quite enough to carry on an efficient circulation.

Some patients without digitalis have a pulse moderate in rate or even fairly slow and suffer no discomfort except some limitation in their response to effort, as in the case of the patient from whom Fig. 130 was taken. The aim therefore should be, as in auricular fibrillation, to give that amount of digitalis which will maintain a moderate rate of the heart, and where such moderate rate is maintained without digitalis, not to interfere. The beneficial effect of digitalis is illustrated in Observation 35. If fibrillation follows the administration of digitalis in flutter, the drug should be stopped. When auricular fibrillation becomes otherwise established the treatment which is suitable to this condition should be followed.

As any matter which throws light upon this condition and upon the action

of digitalis is of value, certain anomalous cases often afford an insight which cannot be obtained from other sources. One patient, to whom reference has already been made, could stop his attacks by producing vomiting, and I got a great number of records from him, and there was no doubt the vomiting often resulted in a stoppage of the attacks. One could reason with fair assurance that this was produced through the action of the vagus nerve, but there was another incident which confirmed this view, where by the sucking of air and belching it there was produced temporarily a marked slowing of the pulse (Fig. 167) without in any way affecting the auricle.

The point I wish to bring out is that this slowing of the ventricular rate is brought about by the action of the vagus upon the a.-v. node, just as happened in the slowing of the rate in Figs. 159 and 160, and that it is through its action upon the vagus nerve that digitalis acts in producing such changes.

The danger of digitalis in auricular flutter.—In three of my cases, after digitalis had restored the normal rhythm, death resulted from cerebral embolism. Manifestly clots form in the auricle during flutter as they do during fibrillation. Contractions of the auricle during flutter are so ineffective that they never empty the auricle of its contents. The sudden resumption of the normal contraction expels these clots, which, reaching the brain, induce attacks of cerebral apoplexy.

Dr. Orr informs me that one of his patients, who had intermittent attacks of auricular flutter, suffered from embolic infarcts in the brachial artery and other vessels on three occasions on the cessation of the attacks of flutter.

The fibrillation that follows auricular flutter.—Some remarkable features about these observations have already been commented on. But there is another which it is impossible at present to explain, namely, the action of digitalis in causing flutter to change into fibrillation, and still more remarkable, the sudden resumption of the s.-a. node in the control of the heart-beat. It would seem as if the auricular fibrillation produced in this way is, in some respects, different from that which ordinarily occurs. I had hoped at one time that this result of digitalis in restoring the normal rhythm would happen to ordinary cases of auricular fibrillation. Although I have tried in many cases to obtain this result, and have pushed the drug as far as I dare, I never had a single success. The reason for this curious reaction so far baffles explanation.

OBSERVATION 38.—Male. Aged 27. Consulted me on March 7, 1910, complaining of sudden attacks of palpitation. The patient had scarlet fever followed by rheumatic fever in 1887, and he had another attack of rheumatic fever in 1902. It was shortly before this attack that he had his first attack of palpitation. The patient was spare, with cold hands and blue fingers. The lips were cyanotic, and his face became very blue on exertion or during an attack of palpitation. The pulse was regular, 64 per minute, and the jugular pulse was of the normal auricular type. The heart was not enlarged, and the first sound was muffled when he stood up.

The attacks of palpitation of which he complained were at first of only a few minutes' duration, and occurred infrequently. During the last few years they had occurred every few weeks and lasted from half an hour to six hours, and one attack, seven years before, lasted a week. The attacks begin suddenly, and he feels weak and disinclined for exertion while they are on, though

he can walk quietly without distress. Violent exertion will bring them on, and also when the stomach feels upset they are readily provoked. As a rule if he takes ipecacuanha wine the attacks will cease a few minutes after he is sick and vomits. He had gone through a great variety of treatments, including a rest cure, but nothing prevented the attacks or cut them short except the ipecacuanha after it made him sick.

At my request he kept a diary for nearly a year, and the following account gives an idea of his experiences for a month:

May 10. Palpitation came on at 10 P.M. Took Ipec. about 10.10 P.M., sick, no effect. Ipec. again at 11.15 P.M., sick, and attack stopped at midnight.

May 17. Woke with palpitation at 5.15 A.M. Took Ipec. twice, sick, and attack stopped at 6.30 A.M.

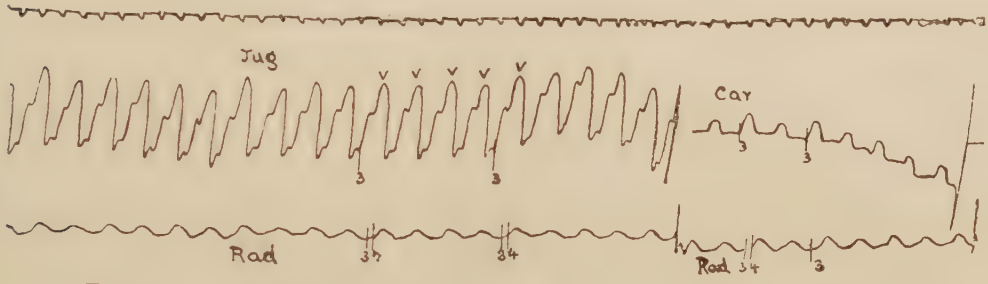


FIG. 165. Tracing taken during an attack of auricular flutter. The jugular pulse is of the ventricular form. (Observation 38.)

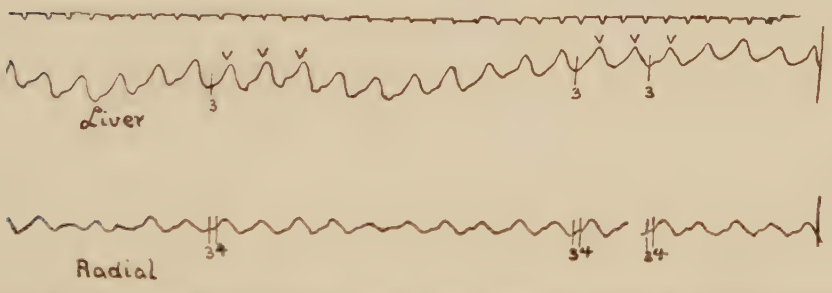


FIG. 166. Tracings of the liver and radial pulses during an attack of auricular flutter. (Observation 38.)

May 22. Palpitation came on when called at 7 A.M. Took Ipec., no result. Had breakfast, took Ipec. again, no result. Finally after having taken four lots of Ipec. the doctor gave me some soup, sick again, and attack stopped at 11 A.M.

May 29. Palpitation came on at 7 A.M. Took Ipec., sick, and attack stopped at 7.40 A.M.

The patient went on in fair health, interrupted occasionally by the attacks of tachycardia, till the middle of January 1911, when he had an attack of rheumatic fever. The temperature was not high, and only a few joints were affected. On the 19th he started an attack of paroxysmal tachycardia which continued till the date of my visit to him on the 24th. He had on several occasions taken ipecacuanha and vomited, but this had failed to stop the attack. When I saw him he was sitting up in a chair, unable to lie down, the breathing laboured; the face, lips, hands, and feet were deeply cyanosed. The radial pulse was frequent and scarcely perceptible (see Fig. 165). The heart's beat was forcible, particularly under the left nipple, and this impulse could be felt as far out as the anterior axillary line. The sounds were clear and distinct. The veins of the neck were full and pulsated (Fig. 236). The liver was 2 inches below the ribs and pulsated (Fig. 166). He was desirous of trying the ipecacuanha again, but his medical attendant

was afraid that he was so exhausted that he would not be able to stand the strain of vomiting. As he felt that he would get relief, and as I had nothing better to offer, I consented to his taking the ipecacuanha again. In half an hour he vomited, and the heart at once reverted to the normal rhythm and he experienced great relief. After this he gradually improved, but the attacks kept recurring with exhausting frequency. I saw him again on February 17, 1911, when he was in an attack which had been going on for several hours. He said he could stop the attack by swallowing and belching air, and this he did, but what happened is shown in the tracing (Fig. 167);

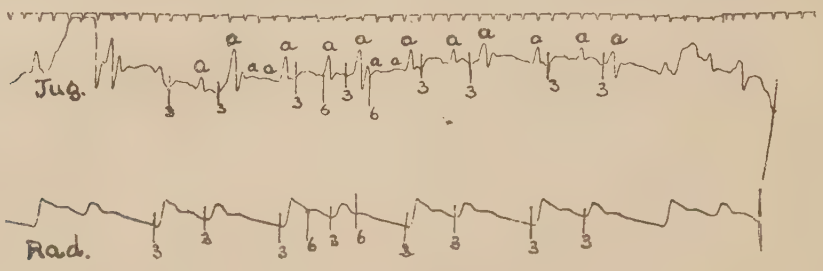


FIG. 167. Shows the effect of swallowing and belching air during an attack of auricular flutter. The pulse changed from that shown in Fig. 165 to that shown here. (Observation 38.)

he probably induced a slight decrease of the excitability of the a.-v. node, so that the ventricle responded less readily to the auricular beats (assuming that it was a case of auricular flutter). After a few minutes it started off at the rapid rate. He was conscious of the temporary character of the altered rhythm, and recognized that it was different from the normal action of the heart. He repeated the experiment and got a similar response. From this date he gradually improved, and for the following two years he was able to follow his profession as a lawyer, though liable to be crippled at intervals by a short attack. In December 1914 he reported that the attacks recurred once a fortnight, and ipecacuanha taken till he vomited usually stopped them. He died during an attack in 1919.

CHAPTER XL

Decreased Excitability of the Genetic System (Heart-Block)

Introduction.
Definition of heart-block.
The inter-systolic period.
The vital process concerned in delaying the passage of impulses.
The *a-c* interval.
Partial heart-block.
Complete heart-block.
The effect of the ventricular systole on the auricular.
Conditions inducing heart-block.
The effect of effort.
The effect of atropine.
The size of the ventricular contraction in heart-block.
Sino-auricular block.
Bundle block.
Arborization block.
Heart-block associated with other diseases.
The age incidence.

Introduction.—It has been shown that when one part of the genetic system becomes more excitable than the s.-a. node, the impulses then start at the excitable part, producing the extra-systole or paroxysmal tachycardia. When the opposite condition happens and one part ceases to function, the part of the genetic system connected with the muscle of the ventricle takes up the function of discharging impulses which results in the contraction of the ventricle.

So far as we understand the matter at present there are only two places recognizable where the stimulus starts the impulses which result in an effective ventricular contraction when the s.-a. node ceases to exercise its function. These are the a.-v. node and the bundle. When the s.-a. node ceases to exercise its function the a.-v. node takes up the function of starting ventricular contractions. When the a.-v. node is destroyed or its connexion with the bundle is interrupted, the contraction starts in a portion of the bundle still attached to the ventricle. The starting of the contraction at the a.-v. node occurs in auricular fibrillation; the starting of the contraction in the bundle occurs in complete heart-block.

This association of auricular fibrillation and heart-block in the same category may seem unjustifiable, but unless the grounds upon which this

association is based are appreciated it will be impossible to grasp the nature of the phenomena presented by these conditions. When their relationship is understood much that is at present confused and inexplicable becomes clear. For instance, the term 'ventricular', or even 'idio-ventricular rhythm', has been applied to the ventricular contractions in complete heart-block. These terms imply that somehow or other the ventricular muscle starts the contraction, whereas, as a matter of fact, it is the bundle that starts the contraction. Again, when that portion of the genetic system situated in the auricle, of which the s.-a. node is a part, ceases to exercise its function the auricle passes into fibrillation, but the starting of a ventricular contraction is not due to the auricle but to the a.-v. node. The auricular fibrillation is a consequence of the cessation of activity of the genetic system in the auricle, and while the fibrillating muscle exercises an influence upon the a.-v. node it is not the impulses from the muscle, but those from the node which cause the ventricle to contract.

This view has led to an intimate study of the a.-v. node, and in the course of this it has been found to be an organ with functions similar to that of the s.-a. node, but with peculiarities of its own. A similar study shows the independent bundle to have functions resembling the s.-a. node and the a.-v. node, but also with individual peculiarities of its own.

In the normally acting heart the peculiarities of the different portions of the genetic system are lost in the harmonious working of all the parts concerned in the heart beat, the variations in rate and rhythm being entirely dependent upon the s.-a. node. There has been already discussed an increased excitability of different portions giving rise to extra-systoles and paroxysmal tachycardias, and the loss of the controlling influence of the s.-a. node, giving rise to auricular fibrillation and auricular flutter.

Portions of the genetic system are liable to a decreased excitability and a cessation of activity. To these conditions the term heart-block has been applied.

When Tawara described the Reizleitungssystem the problem of heart-block seemed about to be solved and it appeared to be a subject easy to comprehend. But continued investigations have revealed phenomena which cannot be explained so long as the system is looked upon as merely one for conducting impulses. A better idea of the nature of these phenomena is obtained when it is recognized that the genetic system has other functions than conduction, and that its conduction is carried out upon a plan different in some respects from that of the nervous system.

Heart-block is, comparatively speaking, a rare condition, but it is a subject of considerable importance in that it throws light upon vital processes which are present in every organ. I have only been able to see a little way into the matter, and I therefore deal with the subject briefly, calling attention to some of the more striking phenomena, leaving to others its further investigation. I endeavour to provide a series of observations on patients which may help other investigators, as few people have the opportunity of seeing a sufficient number of cases which illustrate all phases of the subject.

Definition of heart-block.—By the term heart-block is usually meant an interruption of the impulses which pass from the s.-a. node to the auricle or to the ventricle. But the conception of the term has to be reconsidered, inasmuch as it was given at a time when the impulses were supposed to be conveyed as they are by the fibres of a nerve, whereas they are conveyed in a different manner, described on p. 100. In the meantime the term is retained.

The evidences of this form of disturbance are to be found only in the relation of the contraction of the auricle to that of the ventricle, the auricle being taken as an evidence that the s.-a. node has thrown out impulses, and the contraction of the ventricle as an evidence that these impulses have reached the ventricle.

The inter-systolic period.—In the normal heart, between the contraction of the auricle and the ventricle there is a slight delay—the inter-systolic period. This delay is due to the time taken by the impulse from the sino-auricular node to pass through the a.-v. node and bundle and its branches to the ventricle. As we have seen, the passage of the impulse is from cell to cell of these structures, each cell being stimulated to contraction and discharging at the same time an impulse until the cells immediately in contact with the muscle fibres discharge an impulse which causes a contraction of the muscle.

This process of cell stimulation is dependent upon the excitability of the cell, and if the excitability of the cells of the a.-v. node and bundle are increased, then the inter-systolic period may be shortened; while if the excitability is decreased the inter-systolic period is lengthened, and to this the term heart-block is given.

The vital process concerned in delaying the passage of impulses.—Recognizing that the passage of impulses takes place in the peculiar manner of one cell discharging an impulse which stimulates its neighbour to contract, it seems possible to recognize what it is which causes the lengthening of the inter-systolic period. The prime factors seem to be a delay in the recovery of the cells after discharging their energy. The impulse must reach the cell, and the response cannot be as instantaneous as usual.

According to the law of fluctuation, at no given time are all the cells in activity, so that at one time a certain number discharge their function, and another group at another time. The delay seems to be due to a diminished number of cells being available, so that there is not enough of them to provide a successive relay of cells. The calling upon cells before they have time to recover may gradually lead to extreme exhaustion, and for a time to a suspension of function.

This somewhat crudely expressed proposition is not a matter of mere academic importance—it is necessary for the recognition of a number of processes which we can only indicate by such vague terms as exhaustion, weakness, and the refractory period. In partial heart-block the opportunity is afforded of getting closer to the actual process which brings about such phenomena.

The a-c interval.—The evidence of the duration of the inter-systolic period is found in tracings from the movements of the neck. Waves due to the contraction of the auricle are found in the jugular vein, while the carotid pulse gives evidence of the contraction of the ventricle. These vessels lie close

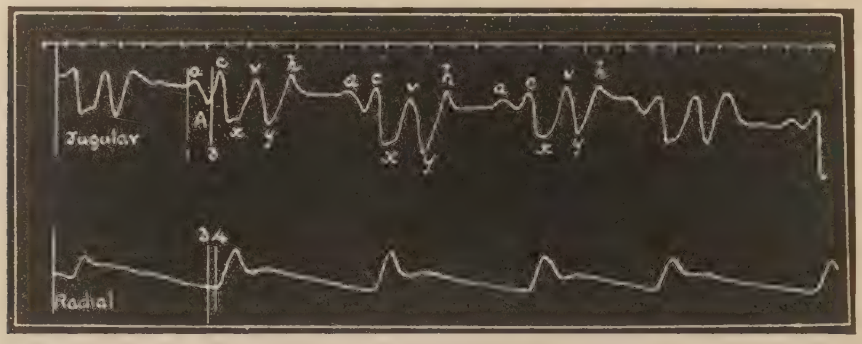


Fig. 168. Shows the duration of the normal *a-c* interval (space *A*) to be $\frac{1}{5}$ of a second in duration. The time marks are $\frac{1}{5}$ of a second.

together, so that by means of a cup or receiver placed over them at the root of the neck a record can be taken by the polygraph. Such a record is shown in Fig. 168, and the inter-systolic period is represented by the interval between *a* and *c*. Normally, this interval is $\frac{1}{5}$th of a second, but it may be decreased or increased. The whole of this interval does not represent the inter-systolic period, because $\frac{1}{10}$th of a second is taken up while the ventricle is raising the pressure to open the aortic valves—the pre-sphygmic interval. As this latter interval is constant any variation in the *a-c* interval can be attributed to variations in the inter-systolic period.

Partial heart-block.—The earliest sign of an interference with the passage of the impulse from the auricle and the ventricle is an increase in the *a-c*

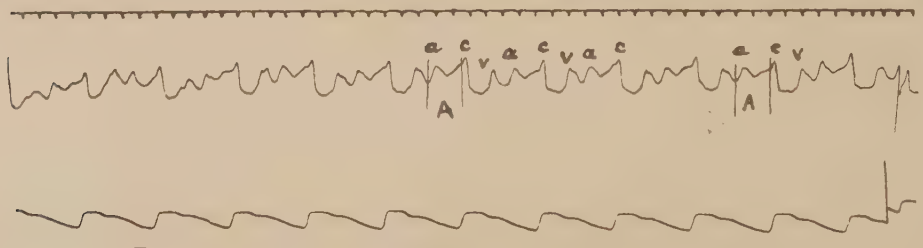


Fig. 169. Shows the *a-c* interval (*A*) increased to $\frac{3}{5}$ of a second.

interval (Figs. 169 and 170). This interval may lengthen until a ventricular response does not occur, and this is shown by the dropping out of a ventricular beat. When this happens the *a-c* interval is continually varying (Fig. 171). After the beat drops out the interval before the next beat may be about normal in duration, but before the following beat it is slightly increased, and it goes on increasing until another beat drops out.

This dropping out of beats may be frequent, so that every third or second beat may fall out, and there may be periods when two or three or more beats may fall out.

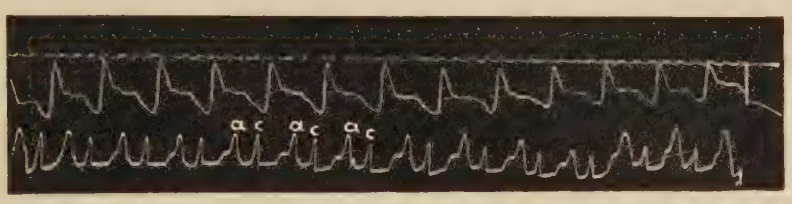


Fig. 170. Shows the *a-c* interval increased to $\frac{2}{5}$ of a second.

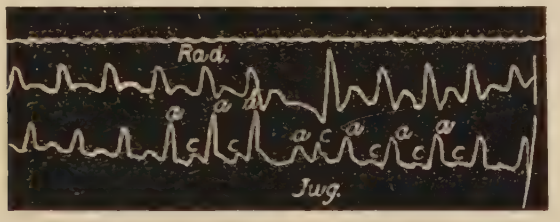


Fig. 171. Shows a gradual lengthening of the *a-c* interval till the stimulus from the auricle at *a'* reaches the auriculo-ventricular bundle before the latter has recovered from the previous stimulation, and finds it 'refractory'. The ventricle, therefore, does not respond to this stimulus, but remains quiescent till the next physiological stimulus comes from the auricle, and the energy of the bundle being restored, the ventricular beat (*c*) follows the auricular wave at a shorter interval. Note the increasing size of the *a* wave before the intermission. This is due to the auricular systole falling at the same time as the preceding ventricular systole, so that the auricular contents cannot be sent into the ventricle, but a bigger wave is sent back into the veins.

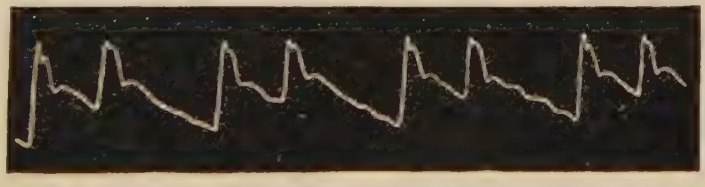


Fig. 172. Regularly intermitting pulse due to partial heart-block. (Observation 7, 1898.)

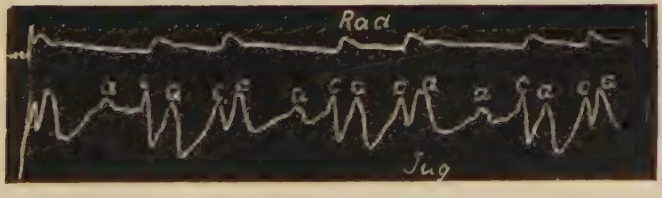


Fig. 173. Taken at the same visit as Fig. 172, and shows the venous pulse during the arrhythmia. The wave *a* is quite regular in its appearance.

Such variations are shown in Figs. 172, 173, 174, and 175, and it will be seen that the s.-a. node still dominates the rhythm of the heart.

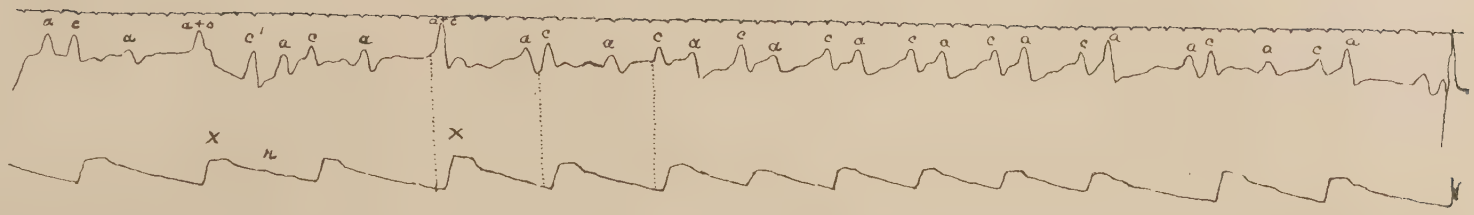


FIG. 174. The irregularity of the pulse is due to periods when every second ventricular systole dropped out, varying with periods when the ventricle responded to each auricular beat. The radial beats (X) seem to be due to a contraction of the ventricle from impulses arising in the a-v node like those in Figs. 53 and 59. If they arose from impulses after the preceding auricular contraction, the a-c interval is unusually long ($\frac{4}{5}$ of a second). The large waves $a+c$ are due to the auricle contracting during ventricular systole. Also shows an extra-systole (c' and r). (Observation 53.)

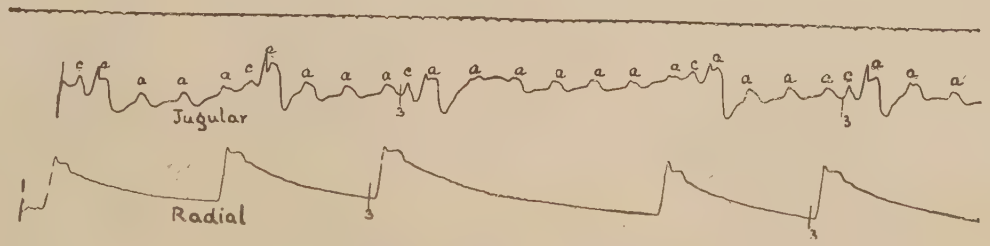


FIG. 175. Shows partial block with a prolonged standstill of the ventricle. (Observation 56.)

Complete heart-block.—In some cases a time comes when impulses fail to pass and the ventricle nevertheless continues to beat, but at a rate much slower than the auricle, and independent of impulses from the s.-a. node.

In such a condition the bundle has taken up the functions of starting the heart's contraction, just as the a.-v. node took up the heart's contraction in auricular fibrillation (Figs. 176 and 177).

Fig. 176. Here the tracing from the neck shows sometimes three auricular waves (*a*) to one carotid wave (*c*). In the last two periods there are but two auricular waves, and the *a-c* interval is longer than in the periods when there are three auricular waves, because in the former case the cell energy has not had so long a time in which to be restored.

This condition is called complete heart-block. The stimulus for the contractions of the ventricle has been supposed to arise in the ventricular muscle, and the term idio-ventricular rhythm has been given on this assumption, but it can be proved that this assumption is wrong and that the impulse for contraction arises in that portion of the bundle which is in connexion with the ventricle. The electro-cardiogram has a definite shape when impulses enter the ventricle by the bundle, and this is retained in complete heart-

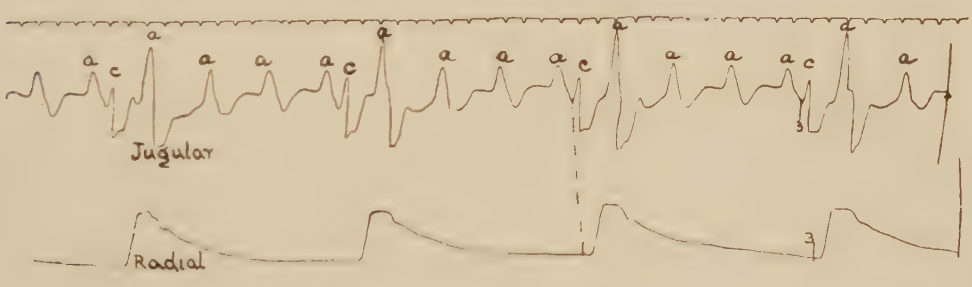


Fig. 177. Tracing from a case of heart-block. There are four auricular beats to one ventricular (4 : 1 rhythm).

block. When the impulses arise within the ventricle the electro-cardiogram has a different shape. Experiment has shown that if the bundle is cut the ventricular contractions are the same as those which occur in the human subject with complete heart-block.

The question may be asked, Is the contraction of the ventricle in complete heart-block not due to impulses arising in the a.-v. node? As we have seen in discussing auricular fibrillation and flutter, the a.-v. node is a peculiarly excitable structure influenced readily by the sympathetic and vagus nerves,

and by other agents. Such agents are entirely without effect in complete heart-block, for it is impossible to increase or diminish the rate by any of those agents which affect the a.-v. node. Thus bodily effort, however violent, produces no increase in the ventricular rate.

The influence of the ventricular beat on the auricular.—On several tracings of 2 : 1 heart-block I have observed that the auricular beat which follows the ventricular systole is slightly premature, shown by the difference in time between the *a-a* intervals. In Fig. 200 it is seen that the *a'-a* intervals are longer than the *a-a'* intervals. This is also seen in the electro-cardiogram Fig. 204. The explanation of this is not yet clear..

Conditions inducing heart-block.—In the majority of cases where there are mild degrees of heart-block the agent merely depresses the excitability of the a.-v. node. They occur in certain infectious or febrile states such as rheumatism, influenza. They also occur in response to digitalis and allied drugs. In most cases the block entirely disappears, leaving no impairment of function behind.

In some rare instances, rare at least in my experience, this functional depression may go so far as to produce complete heart-block. At all events I surmise this from the fact that some patients who show this condition ultimately make a perfect recovery. In others there may be a standstill of the ventricle so long that the patient loses consciousness.

Where there are structural changes affecting the a.-v. node and bundle the early stages are identical with those due to a functional impairment, *i.e.* an increased *a-c* interval, sometimes with the dropping out of ventricular beats. In a number of people the condition goes no further, and patients may be found with a wide *a-c* interval for many years, and occasionally the ventricular beats may drop out. In others the structural changes are slowly progressive until complete heart-block is produced.

The effect of effort.—In complete heart-block bodily effort does not affect the rate of the ventricular beats. I have endeavoured to distinguish between the functional and the structural forms of partial heart-block, but with only limited success. One line of observation pursued was to test the response to effort. As has already been shown, the a.-v. node becomes more excitable during effort, and as a consequence, where the disturbance is functional the heart-block may disappear to a great extent. If there has been dropping out of ventricular systoles, the number that drop out decreases, or they finally cease to drop out.

On the other hand, when there is structural damage to the a.-v. node, effort increases the block, and the pulse-rate falls in response to effort on account of the dropping out of ventricular contractions (see Observation 44).

The reason for this is that the damaged part in the bundle requires a longer time for recovery and is unaffected by effort, so that the impulses arrive at the damaged part before it has recovered.

Unfortunately, since this idea occurred to me I have only had a few opportunities of putting it into practice a sufficient number of times to test

its validity. I put the notion forward here for others to investigate, as it offers an opportunity for a more intimate knowledge of the nature of heart-block and the functions of the a.-v. node.

The effect of atropine.—The atropine test may help to distinguish functional disturbances from structural changes. I have made a good many observations of the effect of atropine in different affections of the heart. To produce a definite effect a dose for an adult is found to be $\frac{1}{50}$th to $\frac{1}{30}$th of a grain of sulphate of atropine injected hypodermically. In some cases of partial block it causes the block to disappear. I had hoped in this way to distinguish between functional disturbance and structural change, but this expectation was not fulfilled, the reason being, I presume, that under all circumstances the vagus action dulls, and its removal increases the excitability of the a.-v. node, both when some of its cells are destroyed and when their function is impaired by some toxic agent.

Some cases of partial block may be due to some agent acting on the vagus, and naturally the suspension of vagal activity at once puts an end to the block.

In complete heart-block I have failed to get any result. There are, however, cases on record where complete block is removed by atropine. This indicates that the lesion could not have been structural, but functional.

The whole subject is still so obscure that it requires a great deal more investigation.

The size of the ventricular contraction in heart-block.—In all cases of heart-block, partial and complete, the ventricular contraction is full and effective. There are no beats like those small ineffective beats of auricular fibrillation, flutter, and the ventricular extra-systole. Though in some cases the damaged bundle must be so greatly reduced in size that only a few impulses can pass through this narrow bundle, yet the ventricle responds with a full and efficient contraction. Thus there must be a stimulation of the whole bundle after the impulses pass through the restricted part. Such a result shows that the intact portion of the bundle which remains in connexion with the ventricle is capable of being stimulated by relatively few impulses reaching it from the a.-v. node. I merely call attention to this fact, as it affords a further opportunity for the study of the peculiarities of the genetic system.

Sino-auricular block.—So far the description has been limited to the decreased excitability of the a.-v. node and bundle. Certain records indicate that the structures, as yet unrecognised, though suspected (p. 90), connecting the s.-a. node with the auricular fibres may also become depressed. This is shown by dropping out of auricular beats, as in Fig. 178. The s.-a. node discharges its impulses at stated intervals to which the auricles respond. When the auricular systole does not occur at the expected interval, it is inferred that the impulse was discharged but failed to reach the auricle. It is not likely that a mere delay or standstill as in Fig. 29 would exactly correspond to two intervals, and be repeated, as in Fig. 178.

Fig. 178 is of interest, as it shows movements due to both right auricle (*a* wave in the jugular) and left auricle (wave *x* in radial tracing), and the absence of both waves during the sino-auricular block (period *o*).

Bundle-block.—One limb of the bundle may be destroyed, and though both ventricles may participate in the contraction they may not be synchronous. This may give rise to the 'cantering' rhythm which is due to the asynchronous closure of mitral and tricuspid valves. There is usually present a wide *a-c* interval.

It is of interest to observe that in this block the efficiency of the ventricular systoles is unaffected, for the pulse-beats are always good and strong. Does the impulse spread from the sound limb to the damaged and stimulate it to a complete contraction as happens in complete heart-block ?

Arborization block.—I doubt if there is such a thing as arborization block. It is an electro-cardiograph diagnosis. It must be remembered that the electro-cardiogram is the expression of the contraction of muscle fibres. If there be an electro-cardiogram which shows an absence of some muscle fibres

Fig. 178. Shows the sino-auricular block. There is an absence of the auricular wave on two occasions in the spaces marked *o*. There is an absence also of the wave *x* in the radial beat. This patient had at the same time partial heart-block, as shown by the absence of the *c* wave and of a radial beat after some of the *a* waves. (Observation 40.)

in the contraction, the morbid condition is in the muscle and not in the branches of the genetic system. This block is said to occur in advanced cases of angina pectoris, but it is in this condition that masses of the ventricular muscle are apt to be destroyed from stoppage of the blood supply (Plate II.). The extent and significance of such changes are better understood from the response to effort.

Heart-block associated with other diseases.—On account of the position of the a.-v. node and bundle, they are liable to participate in diseases that affect the valves, the myocardium, or the blood-vessels.

Mitral stenosis.—The milder degrees of heart-block are fairly frequent in affections of the mitral valve which lead to stenosis. In the majority of cases the heart-block is of a mild degree, leading chiefly to a delay in the passage of the impulse between the auricle and the ventricle, shown by a wide *a-c* interval (Fig. 179). When presystolic murmur has been present there results a mid-diastolic murmur. This murmur is therefore diagnostic of a mild degree of heart-block as well as mitral stenosis. It is in these cases of mitral stenosis with partial block that digitalis acts by producing the dropping out of ventricular systoles. Although I have pushed the digitalis in many of these cases, I have never found it do more than produce a 2 to 1 rhythm.

Aortic valve disease.—Diseases of the aortic valve when they extend into the myocardium may affect the bundle and produce partial heart-block. In my experience this, however, is rare (Fig. 180).

Affections of the myocardium.—It is to be expected that diseases which affect the myocardium would extend to the genetic system. We frequently

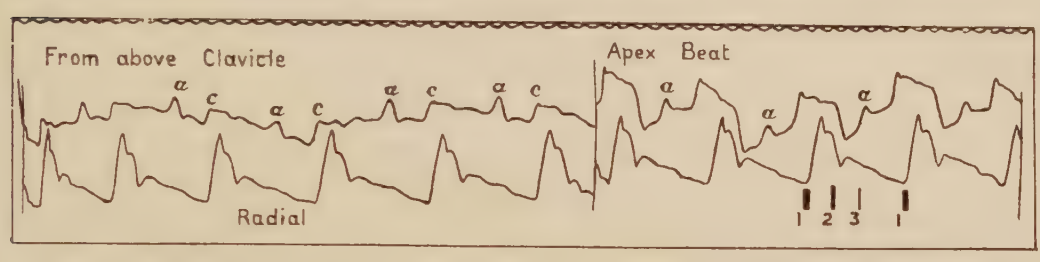


FIG. 179. Simultaneous tracings from the movements in the neck and the radial in the first part of the tracing, and of the apex and radial in the second part of the tracing. The auricular waves (*a*) from the neck and from the apex are seen to be widely separated from the ventricular contraction. The first and second sounds (1 and 2) are represented with a muffled sound (3) at the time of the auricular wave.

find in elderly people with myocardial degeneration a slight degree with partial block, affecting either the bundle or one of its branches (Fig. 181). In the rheumatic heart, mitral stenosis, partial block, and myocardial degeneration may be associated. Extensive disease, such as occurs with gumma of the ventricle, may embrace the bundle and produce partial block. (See Observation 74.)

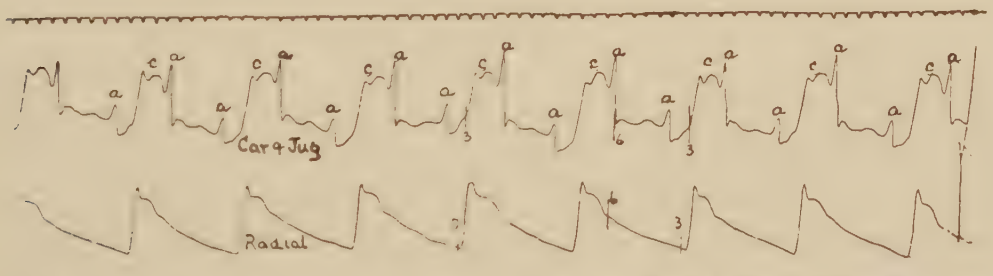


FIG. 180. Tracings from a patient with aortic regurgitation and partial heart-block. The waves *a* are regular, and the difference in their appearance is due to their time relation to the large carotid pulse (2 : 1 rhythm).

Arterial disease.—This, I am inclined to think, is the most common cause of heart-block, being one of the changes which occur in old age, as in Fig. 181.

Auricular fibrillation and flutter.—There are patients in whom auricular fibrillation and flutter may be present as well as heart-block. This association is of some interest, and will be referred to later. The fibrillation and flutter of the auricle have no effect upon the ventricular rate (see Observations 64 and 65).

The age incidence.—Putting aside congenital cases I find in my records two main groups of heart-block due to structural changes where the heart-block is shown to occur as a result of (1) infectious disease of the heart, and (2) senile changes. The former is the result of fibrotic changes following, pre-

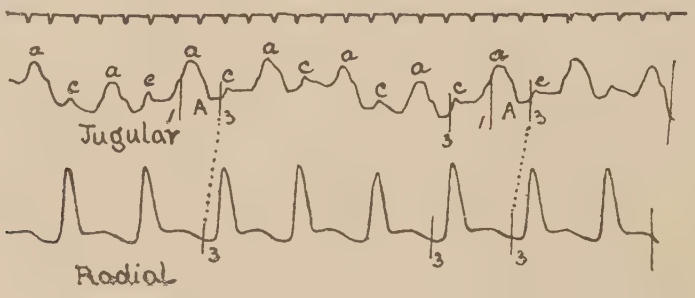


Fig. 181. Simultaneous tracings of the jugular and radial pulses, showing a wide *a-c* interval (space *A*) due to senile cardio-sclerosis. (Observation 34.)

sumably, on an inflammation, as after rheumatic fever, or gummata in syphilis; the latter group results from fibrotic changes due to or associated with disappearance of capillaries. The former are usually mild in degree.

I have constructed a table of my cases of complete heart-block showing the age incidence (Fig. 182). The table is compiled by taking the ages at

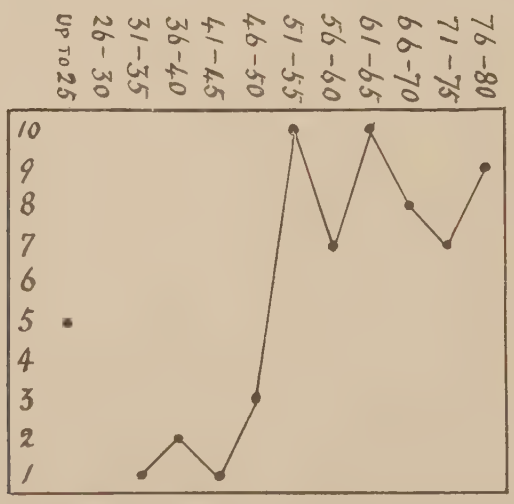


Fig. 182. A chart showing the age incidence of the onset of complete heart-block in sixty-four patients.

which the first evidence of the condition was recognized. Naturally, this is an unreliable method if the object was one that required precision, but as it is mainly to show roughly the period of life in which the condition is most serious and most frequent, it suffices for that purpose.

CHAPTER XLI

Decreased Excitability of the Genetic System (*continued*)

Clinical diagnosis of heart-block.
Heart failure and the symptoms produced by heart-block.
Prognosis.
Treatment.

Clinical diagnosis of heart-block.—Where there is merely a delay in the passage of the impulse and the ventricle responds after each auricular beat, we obtain no evidence of the presence of heart-block by our unaided senses except when mitral stenosis is present, when the mid-diastolic murmur is heard.

When ventricular beats drop out the pulse is intermittent and resembles that when there is an extra-systole whose force is not sufficient to produce a radial pulse. Heart-block may be suspected if there is an absence of those sounds during the long pause which indicates the presence of an extra-systole (see p. 142).

Inspection of the veins of the neck may reveal waves of the jugular vein which are characteristic. If there is an extra-systole the auricular wave falling at the same time may be of large size, and stand out distinctly by itself (Figs. 59 and 61). On the other hand, if the intermission of the pulse is due to dropping out of a ventricular beat from heart-block, the jugular pulse during the pause may be larger, as it has fallen upon the preceding ventricular beat, but the jugular waves before this have gradually increased in size so that it does not stand out as an isolated event, as in the case of the extra-systole (Fig. 171).

When there are longer pauses in the ventricle the persistence of the jugular pulse during the pause is diagnostic (Fig. 175). In complete heart-block the jugular pulse will be found to be more frequent than that of the ventricular beats.

In every case where the pulse-rate falls below 50 per minute the possibility of heart-block must be considered, and the evidences just described should be sought for.

Heart failure and the symptoms produced by heart-block.—Heart failure does not arise from mild degrees of heart-block. Symptoms of heart failure are produced only when the ventricular rate is so slow that the output of blood is defective. These symptoms are exhibited by the organs which receive too little blood to enable them to perform their functions efficiently.

With a regular pulse as low as 25 beats per minute, no symptoms may be produced when the patient is at rest. On effort he may become giddy, or breathless, and these are usually the sensations which compel him to limit his efforts. In some patients the feet feel heavy, and they gradually lose the power of walking at a rapid pace, but can go slowly. The reason for these limitations is that, since the ventricular rate does not increase in response to effort, the supply of blood does not increase to meet the requirements of the body.

When the ventricle stands still over ten seconds, cerebral symptoms dominate the picture. First there is giddiness, then loss of consciousness, and if the standstill exceeds fifteen seconds, twitching of muscles, as those of the face, the neck, and arm, follow (Fig. 183). If the standstill of the ventricle extends over twenty seconds, violent convulsions and stertorous breathing are set up. As soon as the ventricle begins to beat, all these phenomena disappear, the patient quickly recovering consciousness. Sometimes the ventricle beats at rare intervals, in which case the patient has repeated attacks of convulsions and then recovers, when the ventricle begins to beat more frequently. If the standstill is not interrupted, the patient dies.

In one patient who was being operated upon for cancer of the bowel the breathing ceased and the pulse could not be felt. The usual restorative methods were employed without effect, and the patient was laid aside as being dead. Four hours afterwards he was accidentally discovered to be breathing, and the pulse could be felt beating very slowly. The patient did not recover consciousness, and died twenty-four hours later. Here, the likelihood is that the heart did beat at rare intervals.

In the progress of the disease when due to organic changes there is a stage reached before the heart-block is complete when, at one time, the impulses pass through the part that is blocked, and at other times they fail to pass, in which case the ventricle stands still. The resumption of the ventricular beat may be due to impulses getting through again or to impulses that arise in the bundle which is connected with the ventricle. If the ventricle continues to contract in response to impulses from this latter source, then the patient may go on for a long time with a slow pulse, usually about 30 beats per minute, occasionally at 40 beats, or as low as 25. This is the more common result in heart-block due to structural changes, but before the ventricle becomes dependent entirely on the bundle there are periods when impulses do pass through from the auricle and cause the ventricle to contract. These periods intermit with periods when the ventricle fails to respond, and it is during these periods that the attacks of unconsciousness occur.

When the second Stannius ligature is applied to the frog's heart (Fig. 11) so that all communication between auricle and ventricle ceases, the ventricle does not at once begin to beat, there being a pause before this happens. The duration of this pause varies in different hearts. This is what happens in the human heart; and in those hearts where the ventricle does not start beating until after a long delay convulsions and death may supervene.

The dangerous period in heart-block is when it is alternating between partial and complete, *i.e.* between the time that the ventricle is dependent upon impulses from the s.-a. node, and the time that it is dependent upon impulses from the bundle.

In some cases complete heart-block occurs without any previous prolonged standstill of the heart, and therefore without any cerebral signs.

In other cases standstill occurs after the ventricle is regulated by the remains of the bundle, and attacks of unconsciousness occur, during one of which the patient may die.

While the foregoing description accounts for most of the cases of complete heart-block, there are others who begin to have attacks with loss of consciousness, rare at first, and becoming more frequent until complete heart-block is established, whose hearts never show any sign of delay between auricle and ventricle, in records taken by the polygraph or the electro-cardiograph (Observation 59).

The recognition of these cases in the early stage is dependent upon the accounts we get from the patient, or his friends, of his behaviour during an attack, as the doctor seldom has an opportunity of seeing him in an attack. When the patient describes an attack of sudden loss of consciousness, during which he falls down and immediately recovers, the possibility of heart-block should be considered, as such attacks indicate a sudden cessation of blood supply to the brain and its sudden resumption.

There is another class of case in which numerous short attacks may occur, with just a temporary feeling of giddiness, and a sensation as if consciousness was about to be lost. Complete heart-block may even appear for a time and then disappear (Observation 6).

Such varieties of heart-block are, at present, incapable of explanation.

Prognosis.—The mild forms of heart-block are without prognostic significance. In this respect heart-block does not differ from extra-systoles. It is not a condition that materially impedes the

FIG. 183. Is a record taken of one of a great number of attacks of standstill of the ventricle with loss of consciousness. The upper tracing was taken from the receiver of the polygraph placed over the right sterno-mastoid muscle. When the standstill lasted for seventeen seconds convulsive movements were shown in the muscles of the face and neck, and they are represented by the waves in the tracing. They continued till after the first beat of the ventricle. At the same time there were twitching movements of the arm which caused a movement of the pen of the polygraph, giving rise to the waves marked with a X. *y* is the first pulse-beat.

ventricle in its work. When people suffer from heart failure with partial heart-block, the heart failure is not due to the block, but to the conditions that impair the functional capacity of the ventricles, therefore one must look elsewhere for the cause of heart failure. Even when the block is complete and heart failure present, it is not due to the block but to the damaged muscle.

The danger in heart-block lies in the standstill of the ventricle. As has been pointed out, the most dangerous period is when the block is not quite complete, and when the ventricle responds at one time from the s.-a. node, and at other times from the bundle. If this period is tided over, then the patient may live a fairly active life for many years, provided that the ventricle is not damaged, but in some the tendency to standstill occurs and life is always in jeopardy.

Treatment.—Recognizing thus the source of danger, we get indications for treatment, but unfortunately our abilities are not equal to our opportunities. The progressive lesion is beyond our powers to combat (except in rare syphilitic cases), and it remains for us to concentrate our efforts on maintaining the activity of the bundle. I have tried many agents which I had hoped would increase the excitability of the bundle, but all my endeavours failed. Lately some success has been had with adrenalin, which has been found to increase the rate of the ventricle in complete heart-block.[1] An instructive case of complete heart-block, with severe and frequently recurring Stokes-Adams attacks, in which the administration of adrenalin was effective, has been recorded by Phear and Parkinson.[2]

The injection of adrenalin in this case was followed, in fifteen minutes, by complete cessation of the attacks, which formerly had been occurring almost every minute. An interesting feature of the case is that while no change in the complete block resulted from the administration of adrenalin, and no acceleration of the ordinary ventricular rate was observed, the ventricular standstill determining the seizures was apparently abolished by its influence.

This experience is in conformity with a striking experiment by Cullis and Dixon.[3] The bundle in a rabbit was cut and complete heart-block resulted. The injection of adrenalin produced a greatly increased rate of both auricle and ventricle, the latter being independent of the former. The ventricular contractions also were much stronger.

This method of treatment holds out a prospect of success in a condition which has hitherto been beyond our powers to influence.

Milder degrees of heart-block call for no treatment. Where there is reason to suspect a syphilitic lesion or a gumma, anti-syphilitic treatment should be tried.

During the period of liability to attacks of unconsciousness, the patient should be watched and guarded against injury.

[1] Parkinson, J. [2] *Lancet*, 1922, vol. i. p. 933. [3] Cullis and Dixon.

CHAPTER XLII

Cases illustrative of Heart-block

Observations 39-61.

OBSERVATION 39.—Male. Aged 38. He complained of shortness of breath on exertion, and of being conscious that his heart was irregular at times, and easily provoked to rapid action on exertion. He had noticed this first in November 1910, and it has persisted, getting gradually worse, and was very distressing at times. He was in good health until May 1910, when he suffered from slight pain in the right shoulder and elbow, with some grating in the joints on movement. He first thought that the pain was due to playing tennis.

I examined the patient on March 15, 1911, when the pain still persisted in his right shoulder, but he now recognized it as 'rheumatic'. He was pale and the pulse was soft, rapid (104 per minute), with frequent intermissions. The heart's impulse was slight, the dullness extended on the left to the nipple line, the first sound at the apex was slightly muffled.

The examination of the tracing of the jugular pulse showed that there was a delay between the auricular and ventricular contractions, and that the intermission in the pulse was due to the ventricle occasionally failing to respond to an auricular systole (Fig. 184).

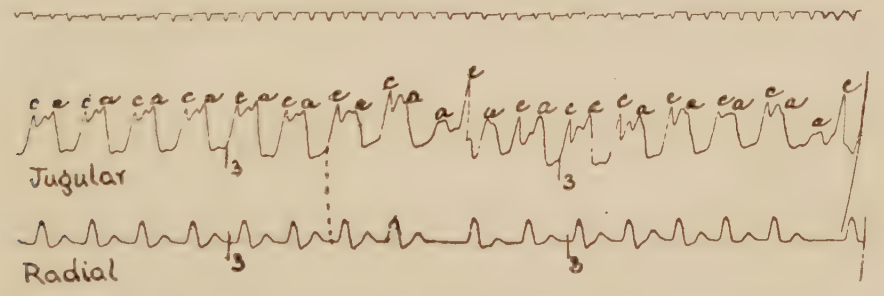


Fig. 184. Shows partial heart-block by the wide *a-c* interval and occasional dropping out of a ventricular beat. (Observation 39.)

An electro-cardiogram showed a marked delay between auricular and ventricular systoles.

The patient was advised to rest, and on examination on the 18th, he showed a perfectly regular pulse, 75 per minute. The tracings from the neck still showed an increase in the *a-c* interval. On April 1 he felt much better; the pulse was still regular at 65 per minute, and the *a-c* interval was of normal duration (¼ second). The patient was advised to continue his work, but to avoid all unnecessary exertion and to rest as much as possible. This he did, and in a few months he felt quite well, and all signs of heart trouble had entirely disappeared. In 1924 he leads an active life with no limitation.

OBSERVATION 40.—Male. Aged 40. Was seized with influenza on March 18, 1902. For five days the temperature varied from 102° to 104° F. After the subsidence of the fever

the patient continued in a weak and prostrate condition, at times mentally depressed and occasionally delirious. Eight weeks from the beginning of his illness he was for several days out of his mind, but finally recovered. During the early periods of mental depression I noticed that his pulse at times became slow and irregular, but nothing abnormal was detected on physical examination of the heart. The pulsation in the jugular vein was at the same rate as the radial pulse when the heart was regular in its rhythm, but more frequent and usually quite regular when the radial pulse was slow and irregular. When records were taken the irregularity was found to be due to partial heart-block. The *a-c* interval being increased when the ventricle responded to each auricular systole, at times the ventricle failed to respond to the auricle, and after such a failure the *a-c* interval became normal in duration (Fig. 185).

In watching the movements of the veins of the neck during the irregular periods of the heart's action, every now and again the veins were suddenly distended by a wave much larger than usual. The tracings (Fig. 38) taken at this time showed that this wave occurred during ventricular systole, and was evidently due to the contraction of the auricle, as the time of its occurrence was that at which auricular systole was due. The auricle and ventricle therefore contracted together, so that when the auricle contracted its contents could not pass into the ventricle, but were thrown back into the jugular vein. This ventricular beat was independent of the auricle, and had its origin in impulses from the a.-v. node. In Fig. 178, which was taken from this patient, on two occasions there is no *a* wave when it was due. There is evidently a block between the s.-a. node and the auricle, for in the spaces marked *o* there are no auricular

Fig. 185. Is from a slow, irregular pulse after influenza, and the jugular tracing shows that the slow pulse is due to the ventricle failing to respond to the stimulus from the auricle. Note that after the short pulse period in the radial, the *a-c* interval is much longer than at the other periods. This is because the fibres have had a short rest, and the conductivity has in consequence not been completely restored. Note also at *x* a slight depression in the radial tracing, due to the systole of the left auricle affecting the arterial column. (Observation 40.)

waves in either the jugular tracing or the radial. It will be seen that in Fig. 185 the small wave *x* in the radial only occurs when there is an auricular wave in the tracing from the neck.

This patient gradually recovered, and for the next fifteen years I kept in contact with him, and examined him from time to time, and could never detect any cardiac disturbance or impairment, and during that period he lived an active life as a sergeant of police.

OBSERVATION 41.—Male. Aged 24. Consulted me in February 1913, because the pulse was always slow. He felt very well and played strenuous games, such as tennis, but when playing hard he got slightly dizzy and mentally confused, and is perhaps more short-winded than other young men. He fainted several times at the age of 17. He had an attack of whooping-cough at the age of 2, and became reduced to a skeleton. He slowly recovered, and the first note made in regard to the pulse was at the age of 5, when a physician found it beating at the rate of 43 per minute. After this date his pulse was always found slow, and he saw a physician at the age of 19, when the rate was still 43. Notwithstanding this he had led an active life. The patient is tall and spare, but healthy-looking. The pulse is regular at 42 per minute. A tracing of the jugular and radial pulses shows that the auricular rate is 87 per minute (Fig. 186). The ventricle is evidently contracting independently of the auricle, that is to say, complete heart-block is present (Fig. 186). The heart is enlarged, the dullness extending $1\frac{1}{2}$ inches beyond the left nipple line. There is a rough systolic murmur heard all over the heart, and I cannot be sure at what region it is heard loudest. I made him run rapidly up and down a flight of stairs and he became slightly

breathless and giddy, but the pulse-rate was not accelerated, and I detected a few long pauses, one of which I caught (Fig. 187). In 1924 he leads a strenuous life, but has occasional attacks of loss of consciousness for a few seconds.

OBSERVATION 42.—Male. Aged 54. A doctor. Seen first 13.6.1917. Complains of being short of breath on slight exertion. Was in good health until 1914, when he was very hard worked.

The patient states that although he has known that he had a heart affection he had been able to lead a strenuous life. His pulse was found to be very slow when he was a child, usually

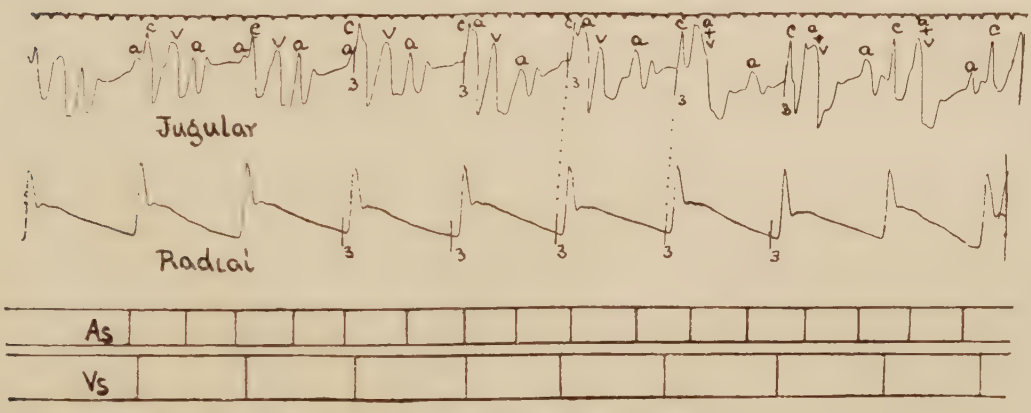


FIG. 186. Tracings from a man aged 24, with complete heart-block—the ventricular rate being 42 per minute. The diagram shows the independence of the auricular and ventricular rhythms. (Observation 41.)

FIG. 187. Tracing of the radial showing a long pause after exertion. (Observation 41.)

between 30 and 40 beats per minute. Nevertheless, he played games like other boys, and when at college played strenuous football, and never suffered any discomfort until during the last three years.

He states that he has had influenza three or four times, and when the temperature is up the pulse-rate may rise to 60 beats per minute, returning to about 36 to 38 when the temperature falls. He is a healthy-looking man although his face is somewhat florid, with a bluish tinge. The pulse-rate is 34 per minute, and the record shows complete heart-block (Fig. 188). The heart is considerably enlarged, extending about 1 inch or 2 inches beyond the left nipple line, and there is a rough systolic murmur heard all over the heart. It is not possible to say in which area it is loudest. On making an effort such as walking upstairs, there is no increase in the rate of the heart.

From this time he continued at work, calling to see me at occasional intervals. He had occasional attacks of bronchitis with cough, and in 1918 he had a bad attack of influenza, but when I last saw him in August 1920 there was no change in the heart. He looked a little bluer, particularly the lips and ears.

He never had any attacks of giddiness or loss of consciousness. When he made an effort he was pulled up by distress in breathing. He had an attack of bronchitis in 1923, and died (aged 60).

OBSERVATION 43.—Male. Aged 59. Seen 18.12.13. Complains of being short of breath for the last five years following an attack of typhoid fever; still he is fit for a good deal

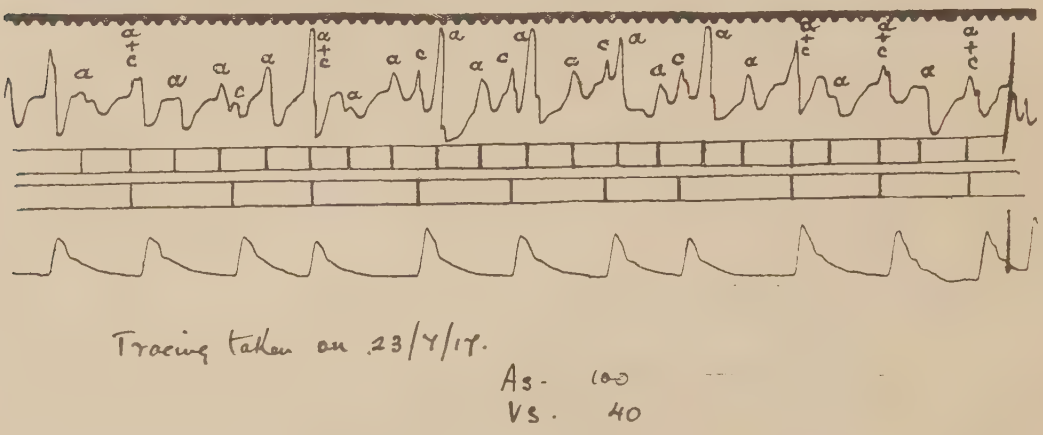


FIG. 188. Shows complete heart-block.

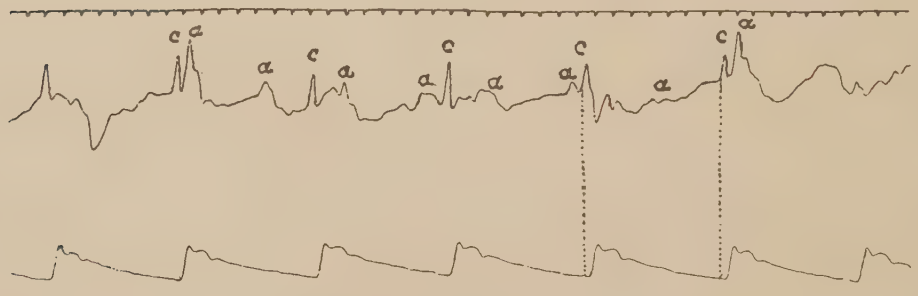


FIG. 189. Shows complete heart-block.

of effort, and can play eighteen holes of golf with comfort. Before the typhoid fever, which was complicated with pneumonia and phlebitis, the pulse-rate used to be about 60 per minute. Of late years it is usually about 40. On examination he was a healthy-looking man—the pulse was 40 per minute, and showed the presence of complete heart-block (Figs. 189, 190). Seen again a year later, the heart was in the same condition, and the rate was slightly greater. In 1921 the condition was still the same. He suffered from repeated attacks of iritis. In 1923 he had an attack of hemiplegia, but all the symptoms passed off in a few days. He reports in June 1924 that he suffers no inconvenience from the heart, but has had to give up playing golf on account of his sight. His pulse-rate in bed in the morning is 40, standing 42. Afternoon, sitting 42. After climbing nineteen steps, 48. He is rather more short of breath on walking. There has never been any syncopal attack.

He is now 70 years of age.

OBSERVATION 44.—Male. Aged 80. Seen March 1914. He felt wonderfully well for his age, but the chief reason for his consulting me was that on making an effort he observed that his pulse-rate, in place of increasing, fell to nearly one-half. I examined him before and after effort, and found that this was true, the rate before effort being 78, and after effort 48. Electro-cardiographic records were taken. Fig. 191 was taken before effort, and shows an auricular and ventricular rate of 78. Fig. 192 was taken after effort, and shows an auricular rate of 92 and a ventricular rate of 46. I again saw him three years later, in June 1917. On the first visit he was able to walk a mile in comfort, but now, after going 200 yards he has to stop because he becomes giddy and feels as if he were going to swoon. If he goes quietly he can get along

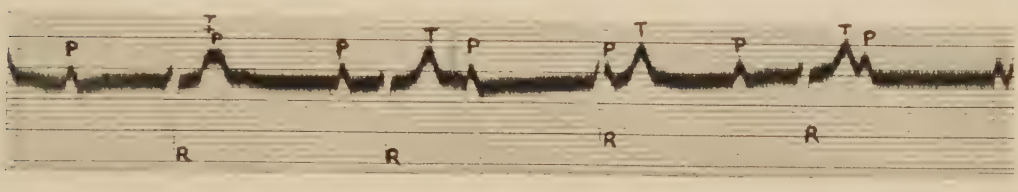


FIG. 190. Is an electro-cardiogram showing complete heart-block.

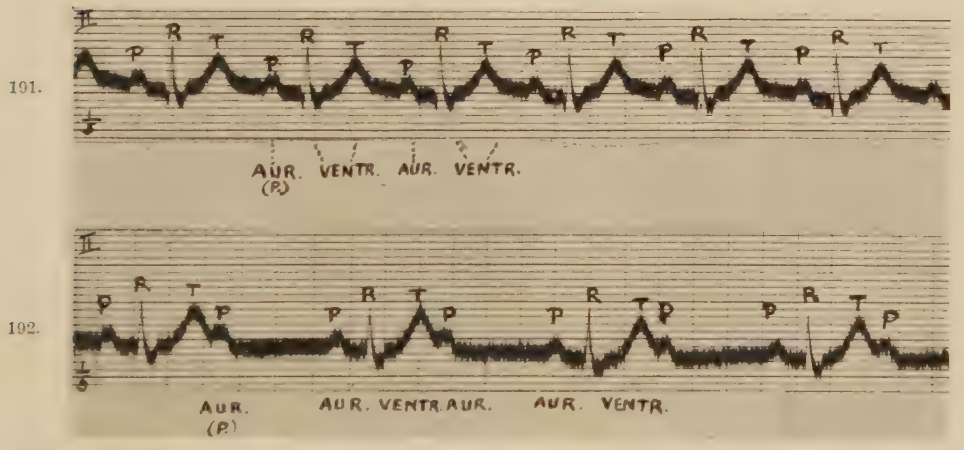


FIG. 191. Is an electro-cardiogram showing partial heart-block. The *P-R* interval is increased. Rates of auricles and ventricles 80 per minute. See next record.

FIG. 192. After Fig. 191 was taken, the patient went up and down a flight of stairs, and his ventricular rate (*R*) fell from 78 to 46, while the auricular rate (*P*) rose from 78 to 92. (Observation 44.)

for half a mile, frequently stopping. If he suddenly stands up from a lying position he becomes giddy. He has twice dropped unconscious in the street and immediately recovered, once two years before and a second time a year ago. He feels giddy before he falls.

He looked well. There were no physical signs except a slight increase in the size of the heart. On slight effort which he made at the examination there was no change in rate.

He states, however, that frequently on setting out to walk, the pulse-rate may be from 72 to 80; in the forenoon, after half a mile's walk, the rate falls to 48 or slower, and may remain at this rate till he goes to bed about 10 P.M. After half an hour in bed the rate suddenly rises to about 70.

I did not see him again, but the attacks of loss of consciousness became more frequent, and

he sometimes hurt himself severely when he fell so that in 1920 he kept his bed, where he spent the last years of his life. His doctor could detect no alteration in his heart.

He became weaker, and his death was sudden at the age of 87.

OBSERVATION 45.—Male. Aged 78. Seen 1.9.14. Has been in good health, and has been used to cycle until lately, when he has become breathless on going up a few steps. He noticed himself that his pulse had become slow a year ago, but it came all right again. A fortnight ago he had a slight attack of loss of consciousness.

On examination he looked healthy; the heart was only slightly enlarged; the pulse-rate was 30 per minute, with a 2 to 1 rhythm (Fig. 193). There was no other sign of heart failure beyond the shortness of breath.

He died suddenly three years later.

OBSERVATION 46.—Female. Aged 60. In 1901 I was asked to see this patient and to state whether she was in a fit state to have chloroform. She suffered from a large tumour in the abdomen. She expressed herself as feeling well, and apart from a little shortness of breath on effort, had no complaint to make. An examination revealed a pulse-rate of about 30 beats per minute, the jugular about double this rate (Figs. 194 and 195). The heart was only slightly enlarged, and nothing further amiss could be detected. The doctor stated that he found the pulse

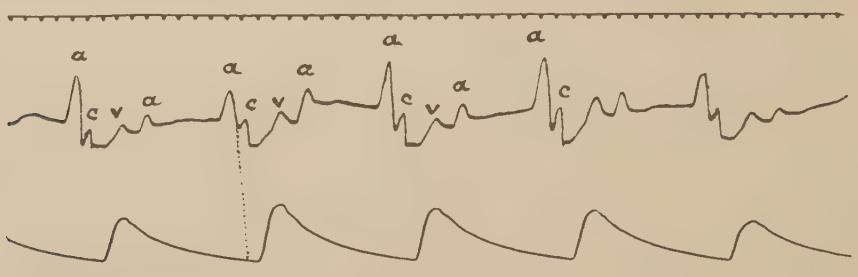


FIG. 193. Shows 2 : 1 heart-block. (Observation 45.)

at this rate ten years before, and it came on, he said, after an attack of influenza. There was no history of syncopal attacks. I advised that she should be operated upon, and I gave the chloroform myself. The tumour was found to be a cyst connected with the liver, and it presented some difficulties in its removal, so that the patient was fully under the anæsthetic for an hour.

During that time I took frequent records of the pulse, and the rate never varied. The patient made a good recovery, and I used occasionally to call and see her. Sometimes she would be resting, but usually she was actively engaged in her household duties, and the pulse-rate was always the same. She was found dead one morning in bed in 1907.

OBSERVATION 47.—Female. Aged 77. Seen March 2, 1911. She has always led an active life, but suffered from colds for the last two or three years.

Five weeks ago she felt tired, and the doctor was sent for and found the pulse at 36 beats per minute. It has remained at this rate ever since. There have been no attacks of loss of consciousness. She is spare but healthy. Pulse about 30 beats per minute, and a graphic record showed 2 to 1 heart-block (Fig. 195). The heart was not enlarged, and the sounds were clear.

From this date she lived a fairly active life for her age, suffering in no way from her heart—neither attacks of giddiness nor loss of consciousness, till 1919. One morning after a good night, she was dressing when she fell down dead (age 85½ years).

OBSERVATION 48.—Male. Aged 76. Seen September 2, 1910. He had been in good health until three years ago, when he began to have attacks of loss of consciousness. The pulse-rate at first used to vary, sometimes becoming very slow. On one occasion it fell to 14 beats, and on that day he lost consciousness forty times. That was two years ago. He has been

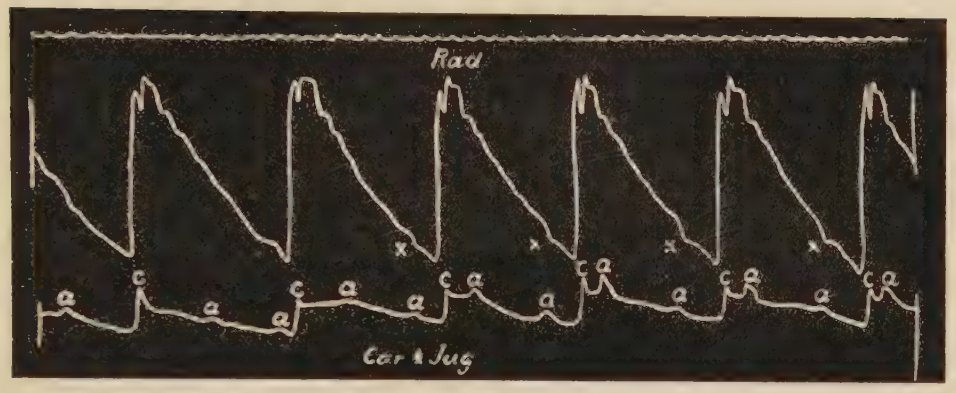


FIG. 194. Shows complete heart-block, the auricle pursuing one rhythm and the ventricle another and slower rhythm. The dip X in the radial tracing is due to the left auricle and corresponds to the auricular wave *a*, so that in this tracing there is a record of both auricles.

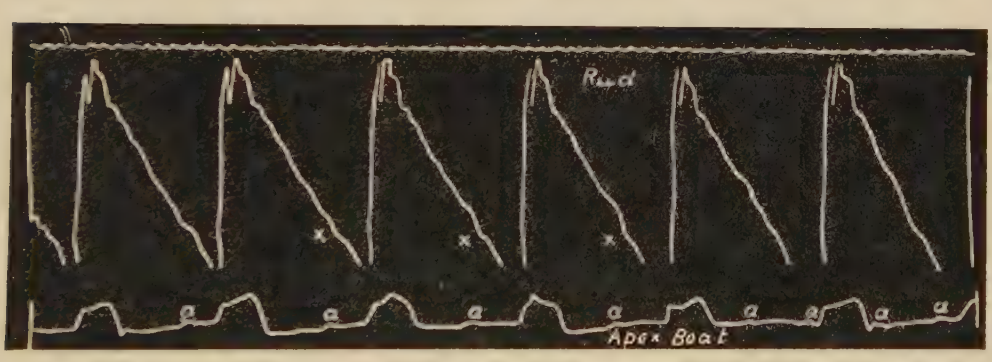


FIG. 194A. From the same case as Fig. 194. Shows the influence of the left auricle on the apex tracing (waves *a*). In the radial tracing the dips (X) due to left auricle are found to occur immediately after the auricular waves in the apex tracing. (Observation 46.)

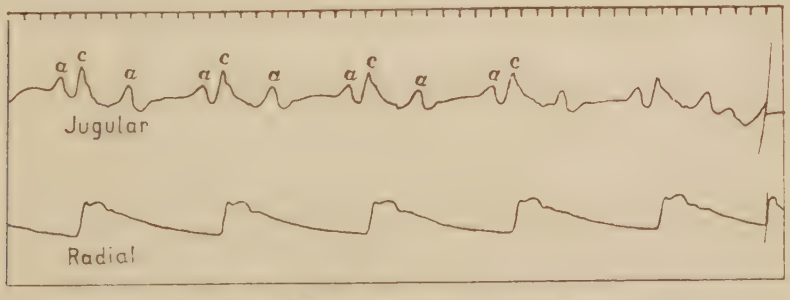


FIG. 195. Shows 2 : 1 heart-block. (Observation 47.)

getting rather weak lately and suffers from pain over the sacrum, and he strains at stool. The motions are loose, and he sometimes loses a little blood. There is a good deal of swelling of the legs, the œdema spreading up into the thighs. The pulse was slow and regular, about 28 beats per minute, and showed a rhythm of 3 to 1 (Fig. 196).

The swelling of the legs had been present for some months, and numerous remedies had been given, including a salt-free diet, theobromine, diuretine, and digitalis—possibly in insufficient doses—with no effect. He was put upon Nativelle's granules, $\frac{1}{240}$ grain, three times a day. He took 14 of these granules, and on the fifth day he became very sick and ill. The digitalis was stopped, and a free flow of urine followed, with disappearance of all the dropsy. He felt extremely well, and went out for a walk which he enjoyed.

He had no more attacks, nor did the dropsy return. The rectal discomfort increased, and it was found to be due to malignant disease, and he gradually became weaker till December 21, 1910, when he had considerable abdominal pain. In the night he became very short of breath and livid, and was relieved by the inhalation of oxygen. He became drowsy, but at 3 A.M. on the 22nd he had præcordial pain for a few seconds, and died. There was no P.M.

OBSERVATION 49.—A distinguished physician who suffered from heart-block communicated to me from time to time his own experiences, and as they contain a description of the great prostration which comes from cardiac weakness, they are well worth recording. He lived an active life, and towards the age of 76 his strength was fairly good for a man of

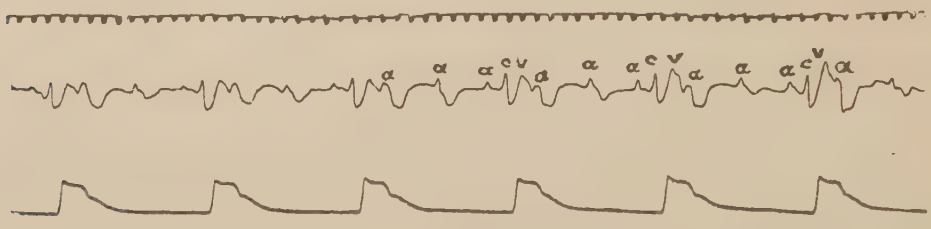


FIG. 196. Shows 3 : 1 heart-block. (Observation 48.)

his years. At that time he began to suffer from syncopal attacks, and for the next two years he had occasionally as many as 20 or 30 in twenty-four hours. His pulse-rate would usually be about 70 per minute, but after a syncopal attack he always found the rate about 30 per minute. At the age of 78 the pulse remained permanently between 20 and 30 beats per minute, and the syncopal attacks disappeared. Two years afterwards he wrote me: 'I am wonderfully free from all the symptoms that usually go along with organic heart disease, my sleep is almost always undisturbed, and I get abundance of it, both by day and night, nor is there the slightest trace of angina pectoris, severe dyspnœa, dropsy, or any of the usual accidents of prolonged cardiac disease.' He wrote later: 'Although a little uncertain in my gait, I can go from one room to another or even up a simple stair, taking plenty of time and assisted by the railing; but for the last two years at least, if not more, my position has been, with few exceptions, recumbent, or at most sitting, and repeated attempts have shown me that it is practically impossible to cross the street or to go into the garden opposite the house except in a wheeled armchair; and along with this there is a feeling of perpetual weariness which never leaves me even after the soundest sleep, and which is not explained by any pain or suffering, though in itself it often tends to fits of yawning and even exclamations which would sound to others as if I was suffering inwardly.'

He died at the age of 82.

OBSERVATION 50.—Male. Aged 48. Seen May 1921. Complains of attacks of loss of consciousness. He states he was quite well till December 11, 1920, when he had a series of attacks of loss of consciousness—about twenty in all. He has had none since, but feels at times as if he were about to lose consciousness. He was kept in bed for two weeks, and was conscious

of the heart beating slow and missing beats. He is able to continue his work, is only slightly breathless, but feels very tired at nights.

Dr. Forrest Brechin's account of his attacks is as follows:

'I was first called to see this man about 9.30 on the evening of Saturday, December 11, 1920, the messenger stating that he was "taking fits, one after another".

'On arriving at his lodgings I found him sitting up in an armchair in a warm room beside a good fire. His face looked pale and puffy with distinct bagginess under the eyes; his forehead was cold and clammy with sweat. He complained to me of feeling very ill and tired, and very cold, and of losing consciousness very frequently since his return earlier in the evening from Glasgow (¾ hour journey by electric tram), where he had spent a quiet time shopping. He said that he had only had one drink—"a glass of the best" at a high-class place of refreshment in Glasgow—and nothing unusual to eat.

'Just as he was relating his past history he said: "I am going off again". He leant back in his chair, his eyes became tightly closed, his face became even paler, and he became unconscious, with increasingly stertorous breathing, and passed into convulsions of face, upper part of body, and upper limbs, with the latter extended and hands clenched. There was little or no movement of lower limbs of themselves. In the course of ten minutes or so he had three of these attacks, and although the attacks only lasted about twenty seconds by my watch, he said on recovering consciousness, which appeared to return more rapidly than it went away, that he felt as if he had been away for a week or more.'

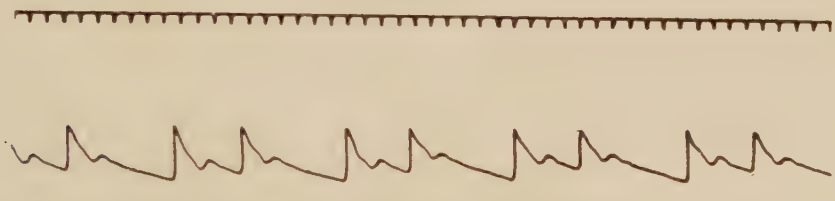


FIG. 197. The pauses in the radial pulse are due to dropping out of ventricular beats. (Observation 50.)

Has had two attacks of rheumatic fever at the ages of 21 and 28. Was refused for life insurance eight years ago. Pulse is 76 per minute, with occasional intermissions (Fig. 197). A tracing of the radial and jugular shows partial heart-block, there being a wide *a-c* interval, and beats occasionally dropping out because of the failure of the stimulus to reach the ventricle (Fig. 197).

The heart was much enlarged, the apex beat being two inches beyond the left nipple line. There was no murmur.

He was given ¼ grain of atropine hypodermically, the pulse-rate being 76. The rate at first slowed slightly, and the ventricular beats continued to drop out for about ten minutes, but in fourteen minutes the rate increased to 90 beats and became quite regular, and the *a-c* interval was normal.

The Wassermann reaction was negative.

In April 1924 Dr. Brechin writes: 'I see the patient occasionally. The rate of the pulse is generally from 45 to 50, sometimes irregular. It has been as low as 36, and as rapid as 80 to 85 per minute. Last September he had a series of attacks of loss of consciousness for two days —about 200 in all. Three weeks later he had another series lasting for one day. He is off work at present since mid-February, because he felt as if the attacks were coming on again.' Atropine was not given during the attacks, as the doctor was away when they occurred.

OBSERVATION 51.—Male. Aged 36. Seen first 1.7.1913. Complains of being easily tired. He cannot walk far or rapidly, as he becomes faint and giddy and feels as if he might stumble and lose consciousness. Talking much will produce the same effect, and he has lost consciousness several times both when walking and talking. When he walks his feet sometimes feel so heavy that he can scarcely drag them along.

He has been delicate since boyhood. At the age of 10 he had a severe illness with a sore throat. He had his first fainting attacks at the age of 21, when he was told that his pulse was very slow. His doctor, writing me, stated that the patient came under his care six years before, that his pulse was 30 per minute, and that he had occasional ' Stokes-Adams attacks '.

The patient gives a clear account of his attacks. Sometimes he knows that an attack is coming on because he is conscious of his heart beating rapidly. Then it seems to stand still, when he becomes very faint and feels that he is about to lose consciousness. Sometimes he makes a violent effort and recovers, but at other times he becomes unconscious.

He is spare, nervous, and delicate-looking. The pulse is 30 per minute, regular except for an occasional coupled beat. The apex beat is well marked, and is felt through the spare chest wall. It is in the fifth interspace, and extends a little beyond the left nipple line. There is a blowing systolic murmur, heard loudest at the apex.

A graphic record showed the presence of 2 to 1 heart-block (Fig. 198).

The condition did not alter for the next few years. In 1916 he began to feel pain in the left chest and arm on walking, but never very severe. In 1917 transient attacks of loss of consciousness became more frequent, and he became more easily tired on walking, talking, or writing. In 1919 he had a series of attacks of loss of consciousness, in one of which he died.

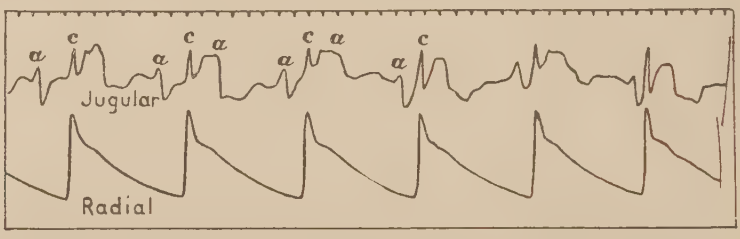


Fig. 198. Shows a 2 : 1 heart-block. The large wave *a* in the jugular tracing is caused by the simultaneous occurrence of auricular and ventricular waves. (Observation 51.)

OBSERVATION 52.—Male. Aged 52. Seen April 1, 1908. Had good health till two years ago, when he observed that he was becoming short of breath on walking to his office. There was also some pain under the left breast, but it was not aggravated by effort. He consulted his doctor and found the heart was acting abnormally. A record taken at the time showed that he had 2 to 1 heart-block.

On examination, he was rather stout. The pulse was rapid, 70 per minute, and the *a-c* interval was normal. The blood-pressure was 120 to 130. The heart was slightly enlarged, extending a little beyond the left nipple line. There was a systolic murmur heard in all the regions of the heart. After this the patient went on very well, but in March and April 1909 the doctor found that he occasionally had periods when the pulse-rate fell to about 40, and tracings showed a 2 to 1 heart-block. In May it had fallen to 32. In June it was sometimes 40 and sometimes 80 per minute, and the rate varied from 30 to 60 till November 1910, when his doctor lost touch with him. He heard later that he died in 1915. He had occasional attacks of ' convulsive seizures ' for two years before his death.

OBSERVATION 53.—Male. Aged 61. Seen January 25, 1909. He was quite well till the last few months. Since that time he has been feeling occasionally giddy, and last week he fainted at the closet.

On examination I find him a healthy-looking man. The heart was not enlarged, and the pulse-rate varied very much, there being periods when the rate was slow and periods when it was more rapid. On a jugular tracing being taken, the slow periods were shown to be due to 2 to 1 heart-block, and there was a wide *a-c* interval during the more rapid periods (Fig. 174). There was a faint trace of albumin in the urine.

Seen nine months later, he states that there have been no more faints, but he felt sometimes very giddy. The pulse now was continuously slow, with a 2 to 1 heart-block (Fig. 199).

Died suddenly in 1913.

OBSERVATION 54.—Male. Aged 56. Consulted me on May 21, 1910, complaining of getting easily tired, especially in the legs below the knees, and of being breathless on the slightest exertion. He was quite well and led an active life till early in 1909, when he observed that he was getting weak and breathless. In January 1909 he felt so weak that he feared he might collapse, and consulted a physician, who diagnosed neurasthenia and heart strain. At this time his heart's action was sometimes slow and the physician said he had bradycardia. He was sent for a long sea voyage, and returned feeling better, but the rate was still slow and he underwent a course of treatment for three months with some slight improvement.

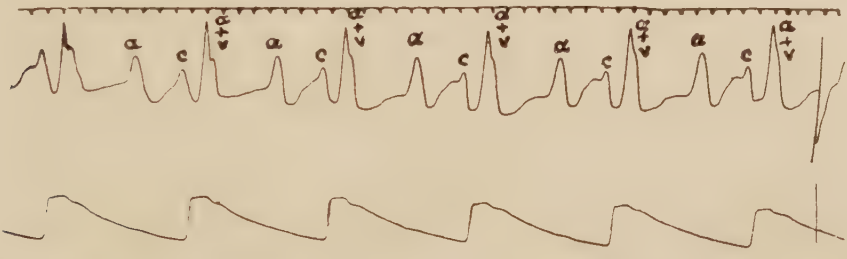


FIG. 199. Shows a 2 : 1 heart-block. The wave $a+v$ is of the same origin as the large wave a in Fig. 198. (Observation 53.)

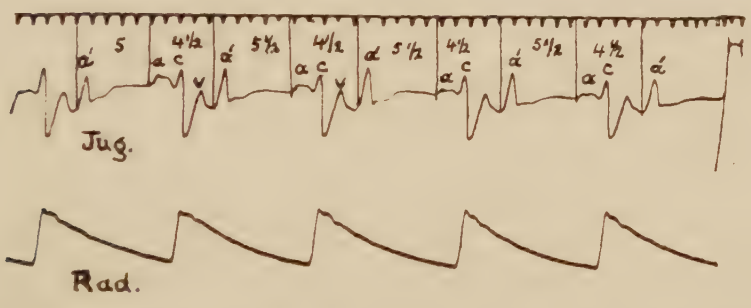


FIG. 200. Shows heart-block when there is but one ventricular contraction to two contractions of the auricle. It will be seen that the interval between the auricular beats varies in that the a interval, which includes the ventricular beat, is less than the other a', due to the premature contraction of the auricle (a') after the ventricular systole. (Observation 54.)

The patient was big and stout, but healthy-looking. The pulse was slow and regular, 30 beats per minute. The heart was enlarged, the dullness extending to the left slightly beyond the nipple line. The sounds were clear. A tracing taken of the jugular pulse showed that there were two auricular waves to one radial or carotid beat (Fig. 200). The auricular beat following the ventricular was slightly premature. The interpretation of the jugular curve was confirmed by the electro-cardiogram. While I was taking a long tracing the patient developed slight Cheyne-Stokes respiration.

Massive doses of oxygen restored the normal rhythm for brief periods, and increased the frequency of the ventricular beats (3 : 2 rhythm) for short periods. During the normal rhythm there was always a wide a-c interval.

On June 26 the patient fell unconscious and recovered after ten minutes. From this date till he died, on July 15, the heart was always slow, and he had frequent attacks of loss of consciousness, sometimes lasting for half an hour, during which time he would have convulsions. The pulse-rate then would be from 4 to 10 beats per minute. During the conscious periods he was very weak and the pulse-rate about 25 per minute, and the auricular rate 90 per minute, the heart-block being complete (Fig. 201). He died during an attack of loss of consciousness with convulsions.

OBSERVATION 55.—Male. Aged 53. Seen March 7, 1917. Two years ago he fell and was unconscious for a short time, and the pulse was found to be about 30 per minute. Up to that time he felt quite well. Two days before I saw him he complained of being dizzy and everything going black before his eyes. The doctor found the pulse about 30 per minute. After

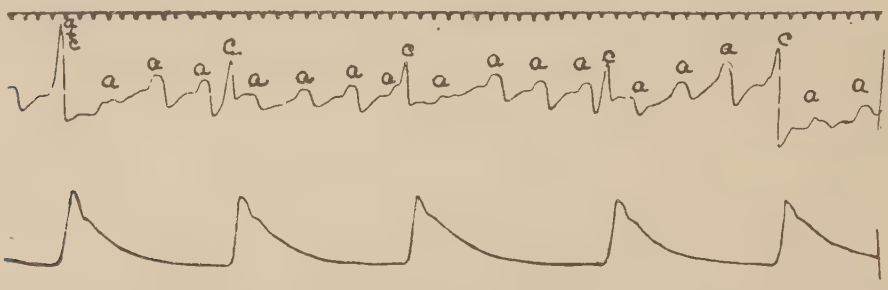


FIG. 201. Tracings from a patient with complete heart-block. The auricular waves, *a*, show a varying relation to the carotid waves, *c*, and to the radial pulse, due to the auricles pursuing one rhythm and the ventricles another. (Observation 54.)

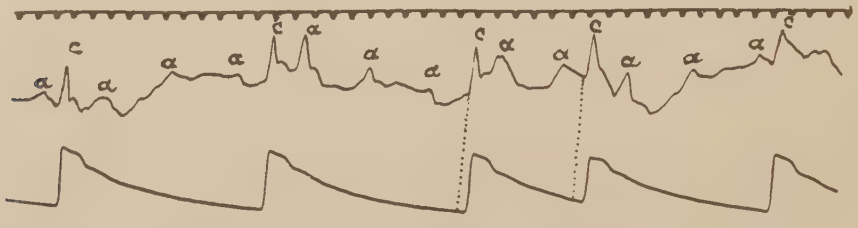


FIG. 202. Shows a 3 : 1 and occasional 2 : 1 heart-block before the onset of attacks of loss of consciousness. (Observation 55.)

this he began to lose consciousness at intervals for brief periods, and sometimes became slightly convulsed. This was the condition he was in when I saw him. He was healthy-looking, well nourished; the pulse-rate about 25 per minute. A tracing showed heart-block (Fig. 202). Every few minutes the pulse disappeared for varying intervals from ten to twenty seconds (Fig. 203). When the rate was about 25, he might be talking quietly when he would suddenly cease talking, close his eyes, lie peacefully, then wake up and resume the conversation. At other times he would become slightly convulsed—his face, hands, and arms twitching for a few seconds.

I saw him again three days later. The condition was the same, only the pauses in the pulse were occurring about once per minute and lasting sixteen to twenty seconds. During the longer pauses he became convulsed (Fig. 183). He seemed to have Cheyne-Stokes respiration, but the pauses in the breathing were found to occur at the beginning of the time when the pulse ceased, and the deep respiration began towards the end of these periods.

During both visits I took continuous tracings of his pulse, on one occasion for 1½ hours

during which time he had lost consciousness fifty times. On March 11 the attacks diminished in frequency, but were fairly frequent for the next three weeks, after which they gradually ceased, and he was able to get up and be about his work, with the heart-rate showing a 2 to 1 rhythm, and sometimes a 3 to 1.

In 1919 he was very well for the first six months. One morning at 10 A.M., in October, he said he felt very well and was about to get out of bed. Shortly after the maid heard him making a choking noise, and when his wife entered the room he was found sitting in his chair, dead.

OBSERVATION 56.—Female. Aged 50. Seen October 18, 1912. The patient had enjoyed good health until five years ago when she had an attack of influenza. Shortly after this she fainted. She also at this time became very short of breath. Frequently since then, upon walking, she begins to feel faint and has to stop for a few minutes, then the attack passes off. Of late she has not been able to walk far because the heart beats violently and rapidly for a few minutes, then it becomes so slow she feels as if it would stop. Ten weeks ago this came on when she was walking. She had to stop, felt very faint, and a cold perspiration broke out, then she lost consciousness.

On examination the pulse was found to be slow and irregular, due to heart-block, the ventricle responding to varying numbers of irregular beats (Fig. 175). The heart was only slightly enlarged, and nothing else could be detected wrong. After this the attacks kept recurring, and she died in one six months later.

FIG. 203. Shows a standstill of the ventricle of 18 seconds. Wave X on the radial tracing is due to a pulse-beat and a convulsive movement. (Observation 55.)

OBSERVATION 57.—Male. Aged 54. Seen first in July 1920. He complains of being easily tired and giddy at times on walking. On walking on the level his feet become heavy, and he is compelled to stop for this reason or because of giddiness. On jumping out of bed he gets giddy and faint, and six weeks ago lost consciousness. On going up a hill he has a heavy feeling across the chest.

He was quite well till eighteen months ago when he noticed that, on lying on the left side, he felt uneasy and uncomfortable, the heart beating heavily and about the rate of 30 beats to the minute. This attack lasted half an hour and recurred for several nights, when it gradually disappeared. In January 1920, after a moderate lunch, he rushed upstairs, and after twenty steps he felt as if he were losing consciousness and sank into a chair which was near. The unconsciousness lasted a few seconds and then passed off, leaving him dazed, uncomfortably weak, and cold with blurred vision. He felt cold for some hours afterwards. A week afterwards he had another attack, and lost consciousness for four or five minutes. The attacks, however, kept recurring for a few seconds, and the doctor who saw him found him pale and ashen in colour with a pulse of 22. He had a few attacks when the doctor was present, and the pulse disappeared preceding the attack. These attacks kept recurring, sometimes with such frequency that he was semi-unconscious. Two weeks later the pulse rose from between 20 and 30 to 66, and the attacks disappeared at the end of January, and now all he feels is occasional giddiness.

He has led a regular and abstemious life, and there is no history of venereal disease and the Wassermann reaction is negative. He looks a healthy man, well nourished. The heart's dullness extends to the left nipple line. The sounds are faint, there is no murmur, and the pulse-rate is about 30 beats per minute. For a short time during the examination the heart-rate increased to 60 per minute.

Polygraphic and electrographic records were taken at various times during the ten days he was under observation. The pulse was usually regular and slow, 30 beats per minute (Fig. 204), but occasionally it would be at 60, the ventricle responding to every auricular beat. The effect of atropine was tested (see below), and the patient was put on the drug—$\frac{1}{100}$ grain three times a day. There was no return of the attacks of unconsciousness for some months, and the pulse-rate varied from 30 to 60 or 70 beats. The atropine was stopped, and no difference was detected the heart-rate varying in the same way. During January 1921 he had one or two slight attacks of loss of consciousness. I saw him again in April 1921. He looked well, and had been feeling well, but in the course of my visit to him he was seized with an attack of pain which I attributed to gall-stone colic. A hypodermic injection of morphine was given, which gave relief.

I did not see him again, but a month after my visit he was seized with repeated attacks of unconsciousness, and passed into a state of coma, during which he died.

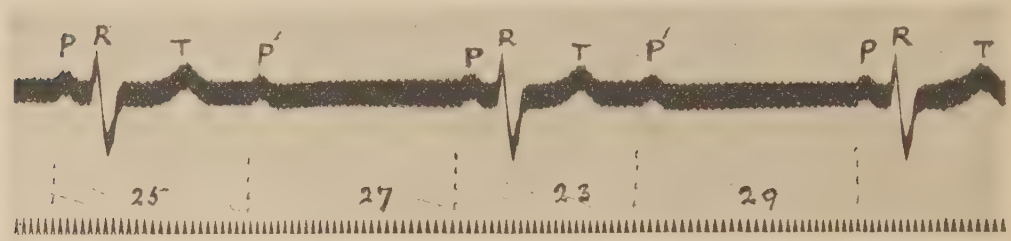


Fig. 204. Electro-cardiogram shows case of 2 : 1 heart-block showing the same features as Fig. 200, the auricular waves *P'* after the ventricular systole being premature.

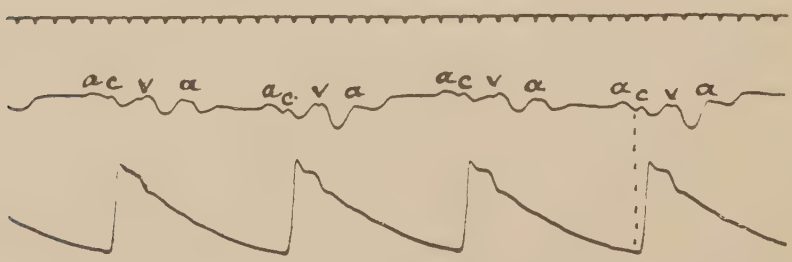


Fig. 205. Shows a 2 : 1 heart-block before the injection of atropine. Auricular rate 60 per minute. (Observation 57.)

At a period when the ventricular rate was half the auricular he was given nitro-glycerine and nitrite of amyl without any change being produced. On another occasion $\frac{1}{60}$ of a grain of atropine was given hypodermically. Fig. 205 is a record taken ten minutes before the injection, and a continuous record was taken for an hour and a half afterwards. An analysis of the records showed no change for twelve minutes, when there began to appear at rare intervals groups of two beats of the ventricle. These became gradually more frequent till thirty-five minutes after the injection, when the ventricle responded to every auricular beat (Fig. 207). This continued for the remainder of the observation. Fig. 208 is the portion taken one hour after the injection of the atropine.

There is shown in these tracings and in the electro-cardiograms no increase of the *a-c* or P.R. intervals. Another peculiar feature is that at no time did the auricular rate increase from the effects of the atropine, indeed it actually became slower, for the auricular rate at the beginning of the observations and for some time after was 60 per minute, while an hour later, when the

block had passed off, it was 55 (Fig. 208), and it was still at this rate an hour and a half later at the end of the observation. The question arises, was the removal of the block not due to the slower rate of the auricles ?

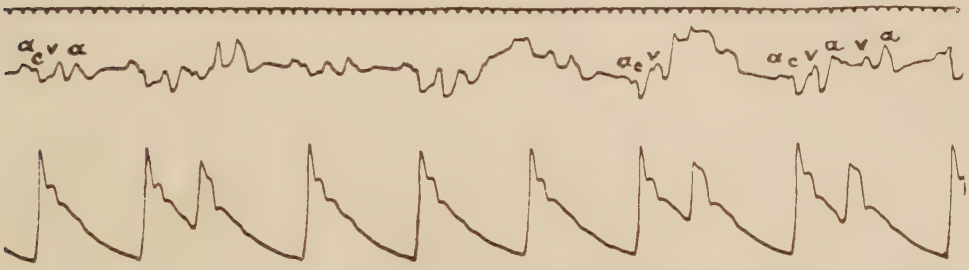


Fig. 206. Fourteen minutes later shows the occasional appearance of a 1 : 1 rhythm.

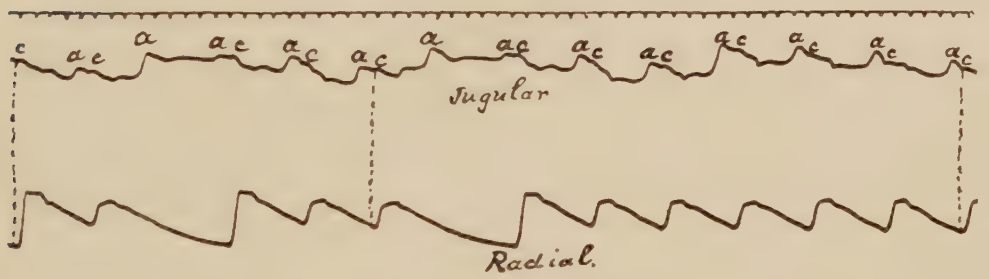


Fig. 207. Shows the disappearance of the block, 24 minutes later.
Auricular rate 60 per minute.

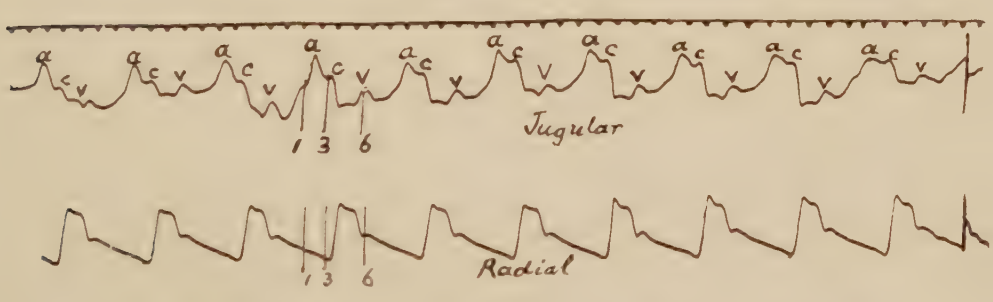


Fig. 208. Was taken an hour after injection, and shows the normal rhythm.
Auricular rate 55 per minute. (Observation 57.)

OBSERVATION 58.—Male. Aged 61. Seen March 6, 1917. The patient is a doctor. He complains of feeling well at times, but is liable to attacks of giddiness. Sometimes these giddy attacks end in a loss of consciousness, which may last more or less for half an hour.

The first attack was in August 1916, when he felt giddy whilst standing still, and fell down and became unconscious for a second or so. He recovered at once. He had another attack four months later.

The next time was on February 17, 1917, when he felt giddy on standing up, and at once lay

down on the floor feeling very faint, and became unconscious for a very short time. He remained on the floor for an hour, and then got up and was all right. From this time he began to have these attacks more frequently. When I saw him he was feeling quite well, and on examination I could detect no abnormal sign. The heart was normal in size, the rate between 70 and 80, and there was no shortening of the *a-c* interval. During his attacks he said that his pulse used to vary remarkably in rate. After an attack he would find his pulse about 26 beats per minute, and while it was so slow he at times felt very faint and giddy, and as if he would lose consciousness. In the morning his pulse was generally about 70 to 80 beats per minute. Towards night it tended to become somewhat irregular, and sometimes fell to 30 or 40 beats per minute.

He wrote to me in September stating that he was having frequent attacks, so that to avoid them he had to lie flat with the head not raised, because if he sàt up or stood up he became very giddy and had attacks of unconsciousness. He dared not sit up until the rate had increased, and when the pulse rises to over 70 he finds it safe to get up. He continued in this way until October of 1917. One day he got up feeling rather well, and was walking to his front door smoking a cigarette, when his wife and son heard a crash, and found him lying just inside the door. He never moved, and when his colleagues saw him a few minutes later there was no sign of life.

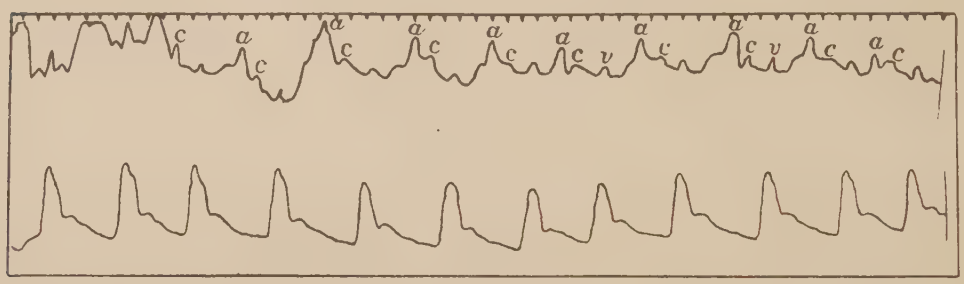


FIG. 209. Tracing showing a normal rhythm at the early period of heart-block attacks. The *a-c* interval is slightly increased. (Observation 59.)

OBSERVATION 59.—Male. Aged 65. Examined November 21, 1919. He complains of having dizzy attacks, and of having lost consciousness for a few seconds on two or three occasions. On one occasion while at his work as a joiner, he suddenly fell and was unconscious for a few seconds, but immediately recovered and went on with his work. Walking along the road one night, a brightly-lit car passed and he fell unconscious, having said Oh! as he was falling, and immediately recovered and walked on.

He complains of giddiness on rising up suddenly.

On examination he presented the appearance of a healthy man. The examination of his heart revealed nothing amiss. The pulse was 58 and quite regular, and a jugular tracing showed no increase in the *a-c* interval. The provisional diagnosis on account of the peculiar nature of the attacks was heart-block.

From this date onwards the attacks came on with greater frequency, usually lasting only a minute or two. His doctor reported on December 14, 1919, that there had been no fainting for a week, but that there had been giddiness. While the doctor was taking his pulse, which was 58, there were at frequent intervals long intermissions, but the patient did not lose consciousness.

Up to 28.12.1919, he had occasional brief attacks of loss of consciousness, and on this date, while having an evening meal, he cried to his wife, then became unconscious. He also became very pale. He fell off his chair, and after a few seconds there were jerking movements of his limbs. He remained unconscious for about five minutes, and was in a dazed condition for some time afterwards. He was also very sick and vomited frequently, and when seen about three hours afterwards by his doctor the pulse was 22 per minute. From this date he had frequent attacks of giddiness and unconsciousness, sometimes with slight convulsions. The pulse was

frequently about 18 per minute, sometimes rising to 24. Gradually the pulse returned to 30 per minute, and he was free from attacks for several days (Fig. 210).

On January 11, 1920, the rate was about 35 per minute (Fig. 211). There was a slight cough, and he coughed up blood-stained sputum. He had no more attacks of loss of consciousness until the 20th, when he complained of great giddiness while lying still. The pulse was found sometimes as slow as 10 per minute, and he then complained of great weariness and exhaustion. During that night his wife was wakened by the patient's heavy breathing. He then had repeated

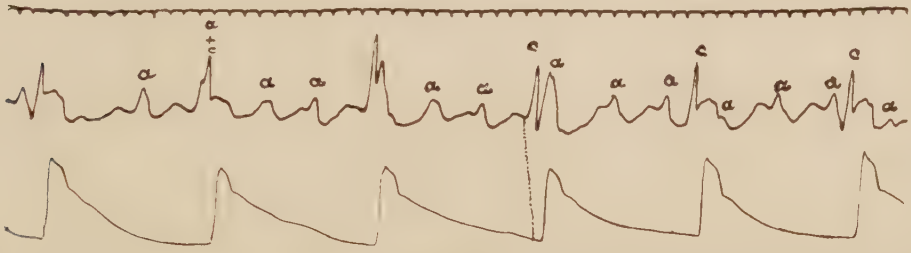


FIG. 210. Shows complete heart-block. The auricular rhythm varies slightly. (Observation 59.)

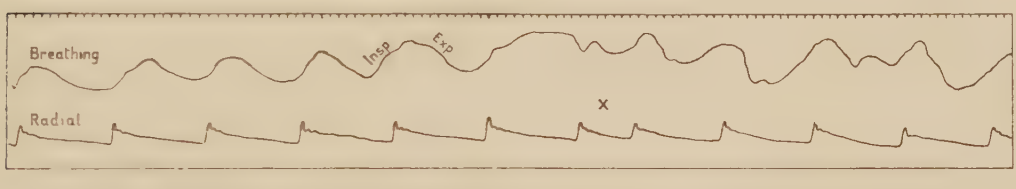


FIG. 211. Shows the pulse and respiration at the same rate. At X a premature contraction occurred, and the respiration was disturbed at the same time.

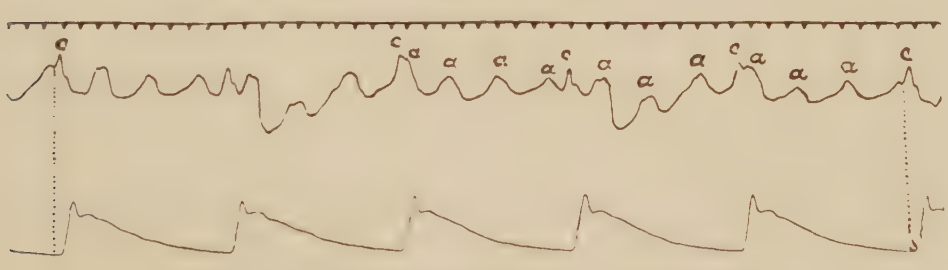


FIG. 212. Shows complete heart-block. (Observation 60.)

fits, breathing seemed to stop at intervals and start as suddenly, and loudly, and when breathing in this manner he was scarcely conscious and was very restless. This condition persisted until noon, when he died.

OBSERVATION 60. Female. Aged 70. Seen March 12, 1914. Was well and active until eleven months ago, when she had an attack of loss of consciousness which lasted five hours. A few days ago she had another attack which lasted the same time, and during that time the pulse-rate was about 30 beats per minute. After her first attack the pulse rose to 80 per minute, and it continued at that rate until lately, when it sometimes fell as low as 40.

When I examined her she was in bed, the face somewhat flushed. She was rather spare. The heart was not enlarged, and there was no murmur and no signs of dropsy. The patient said she felt very comfortable, but every few minutes she felt dizzy. On taking a tracing, the pulse-rate was about 30 per minute (Fig. 212). There were occasional long pauses. It was

then she felt dizzy. These are shown on Fig. 213. I observed while taking the tracings small movements of the lever which was recording the radial pulse during the longer pauses—some of which are indicated in the tracing at X.

Professor MacWilliam, to whom I showed the tracing (Fig. 213), identified the small beats as due to fibrillation of the ventricle.

A few hours after I saw her she became unconscious and died.

OBSERVATION 61.—Male. Aged 47. Seen 24.2.1913. Complains of giddy attacks. The first was seven months ago, followed by another attack a month later, then no more until a month ago, and in one night had several attacks (during the attack his doctor said the pulse disappeared).

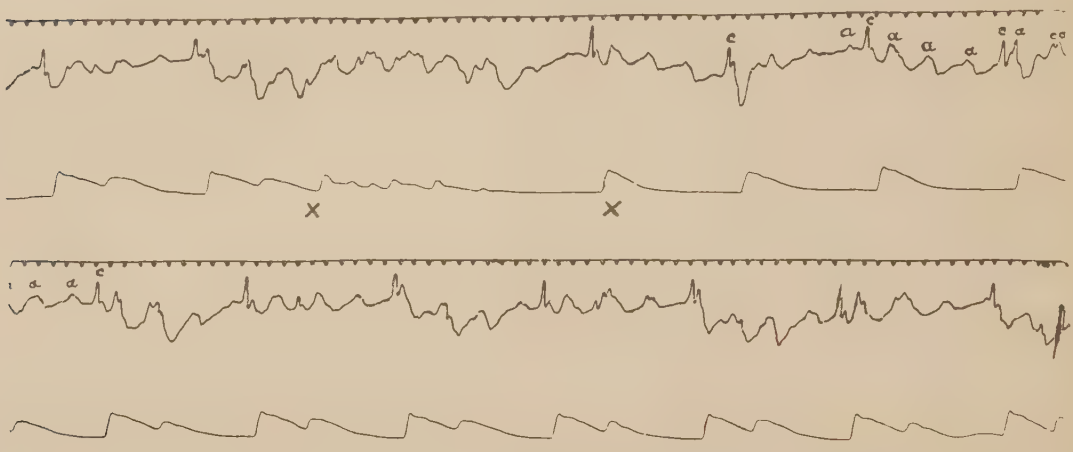


FIG. 213. Shows a period from X to X when the patient partly lost consciousness. There are small waves seen in this record which Professor MacWilliam says indicate ventricular fibrillation. (Observation 60.)

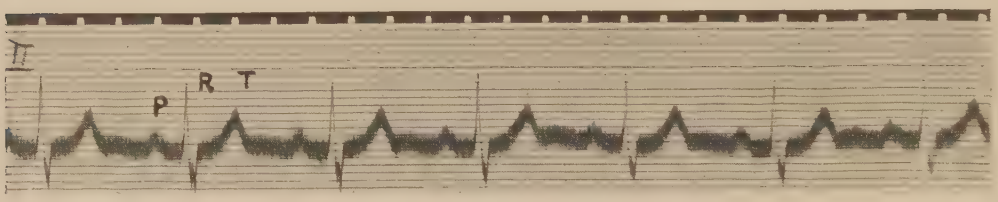


FIG. 214. Shows the electro-cardiogram of the normal acting heart. See Fig. 215. (Observation 61.)

Nine days ago, while standing up after a short walk, he had a giddy attack and fell down and was unconscious for about half a minute. There is no history of any previous illness, and he had considered himself a healthy man, and had no difficulty on walking up hills. On examination the pulse was 60 per minute, and perfectly regular. The heart dullness extended to the left nipple line, and there was a rough systolic murmur heard at the apex and at the base.

While the electro-cardiogram was being taken the first record showed a normally acting heart (Fig. 214), but while the second record was being taken the patient had one of his giddy attacks and the ventricle had ceased to beat for a short time (Fig. 215).

A tracing of the pulse of this patient during a giddy attack is shown in Fig. 216.

Wassermann reaction negative. He went on very well for a fortnight, when he had another

momentary attack of loss of consciousness. He felt weak and consulted his doctor, who found the heart rapid and irregular. The patient was put to bed, and I saw him a week later, looking very well but nervous. The pulse was rapid and irregular, due to auricular flutter. Two days later his doctor stated that he had had several attacks, during which he became very pale, but did not lose consciousness, and the doctor found that during such an attack the pulse was very slow. I saw him on 31.3.14; his pulse was 50 per minute regular, and a record showed complete heart-block (Fig. 217).

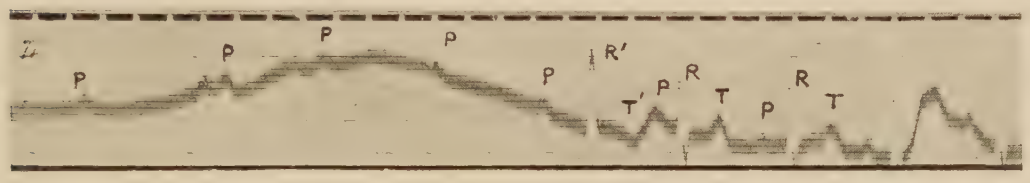


Fig. 215. Was taken immediately after Fig. 214. The patient partially lost consciousness. It is seen that during this period the auricle (waves *P*) continued to beat while the ventricle stood still. The first beat of the ventricle (*R'*) was evidently due to an independent discharge of impulses from the a.-v. node.

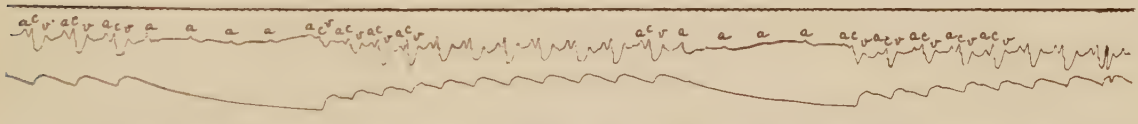


Fig. 216. Tracings of the jugular and radial pulses during two syncopal attacks, due to the sudden cessation of ventricular activity, while the auricle continued to beat. (Lewis.) (Compare with Fig. 215.)

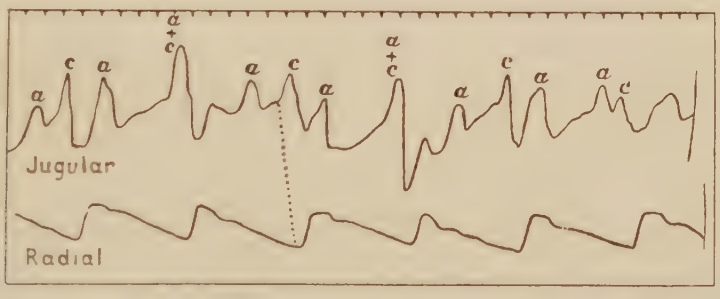


Fig. 217. Shows a complete heart-block with the ventricular rate 50 per minute.

The patient went on fairly well, with occasional attacks of loss of consciousness, until July 1914, when he fell ill with an acute febrile attack which, from a blood culture, was found to be of the nature of streptococcal septicæmia. The illness was a somewhat severe one, but he kept fairly well, although he had an attack of loss of consciousness with convulsions three days after the fever set in. There is no record of the pulse during these attacks, but the doctor states the pulse was usually increased in rate during this illness. There was a good deal of headache present during the illness, with muscular twitchings and sleeplessness. There was no increased reflex nor any Babinski reflex. Two days later he had another attack of loss of consciousness with convulsions, lasting only a few seconds. He gradually sank, and died ten days from the onset of the fever.

CHAPTER XLIII

The Pulsus Alternans

Conditions favouring the appearance of the pulsus alternans.
Clinical diagnosis.
Differential diagnosis.
Prognosis.

This is a form of pulse which is regular in rhythm, but where a big beat and a smaller beat alternate (Fig. 218). Its origin is still in dispute, but I am disposed to include it amongst disturbances of the genetic system.

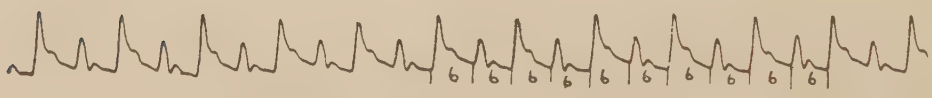


Fig. 218. Radial tracing showing the pulsus alternans. Each period is of exactly the same duration (six-tenths of a second).

In favour of this view is the principle sought to be established in this book that every contraction, orderly and efficient, is the result of impulses conveyed to the muscle by its regulating structure, and just as the hand closes gently or strongly from such impulses so this variation of the heart-beat arises in the same way. By this line of reasoning we infer that the disturbance is situated in the a.-v. node. In favour of this view is the passage in auricular flutter of the pulsus alternans into that peculiar irregularity, to which attention has already been directed (p. 218, and Figs. 7, 8, and 9).

If this is the case then the capacity for inducing the alternation must also be possessed by the branches of the conducting system as it is shown to occur in extra-systolic tachycardia (Figs. 78 and 79). In these tracings it will be seen that the pulsus alternans occurred not only with the abnormal rhythm but also when it became normal.

On this question the electro-cardiograph throws no direct light, for it is only rarely that any sign of alternation is present. On these rare occasions

the small radial beat is accompanied by a smaller *R* wave, from which can be reasonably inferred that the smaller *R* waves are due to fewer fibres contracting—assuming that the electro-cardiogram is the result of the contraction (see p. 426). In some records the reverse is found, the smaller radial being present at the same time as the larger *R* waves.

These remarks are made on the assumption that the pulsus alternans represents a variation in the force of the ventricular contraction and is not due to some mechanical factor. Lately the supposition has been put forward that it may be due to an escape of blood back through the mitral orifice during the contraction of the ventricle—such escape being greater during one beat than during the others.

In such experiments as these of Engelmann on the frog's heart described by Wenckebach, it must be remembered that in interpreting his results Engelmann had no knowledge of the genetic system, and any interpretation which does not take it into consideration is bound to be fallacious.

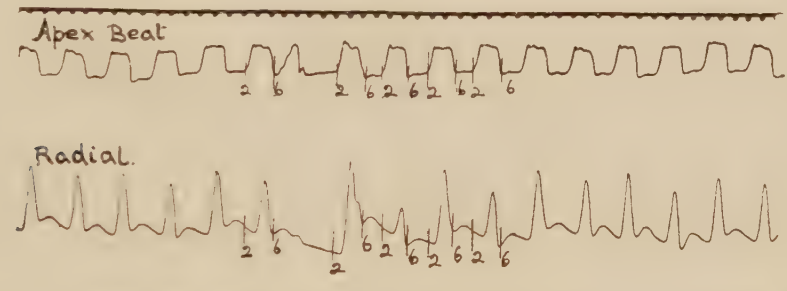


Fig. 219. Simultaneous tracings of the apex and radial. After an extra-systole the alternating character of the radial pulse becomes more marked. Notwithstanding the marked difference in size of the radial beats due to the alternation, there is no difference in the size or duration of the apex beats—in marked contrast to the apex beat caused by the extra-systole.

Conditions favouring the appearance of the pulsus alternans.—There are a variety of such conditions, of which one is the change that occurs in hearts where degenerative changes have taken place and the muscle is replaced by fibrous tissue. While the pulsus alternans is mostly found in the elderly it may be present in people at any age when there are degenerative changes in the heart. In such cases the inference is that the degenerative changes are not limited to the muscle but affect also the genetic system.

It is not always constant, there being a tendency for it to disappear when the patient is at rest, and to reappear on effort. It may also appear for the first time only after an extra-systole. When it is present an extra-systole is always followed by a marked increase in the alternation (Figs. 219, 220, 221 and 222).

There are no doubt other circumstances in which it occurs than with degenerative changes. The late Dr. Gordon Sanders showed me a record of the pulsus alternans taken from a man 50 years of age during an attack of

renal colic. It was never found at any other time, and the patient at the age of 63 continues to lead a healthy life.

These remarks apply to patients with a normal rhythm. There is a

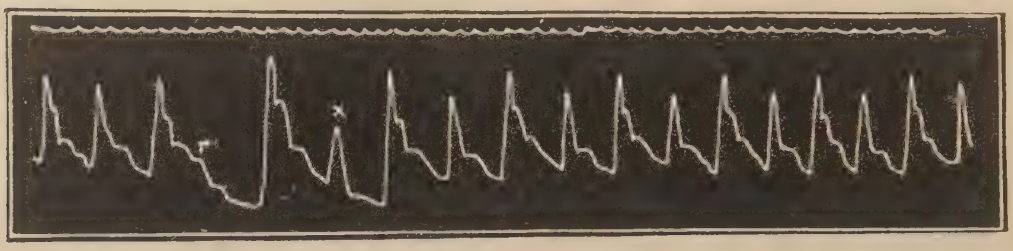


Fig. 220. The alternating character of the pulse is increased after the long pause following the extra-systole (r'); the second beat (X) following the pause appears at the normal interval, but is greatly reduced in size.

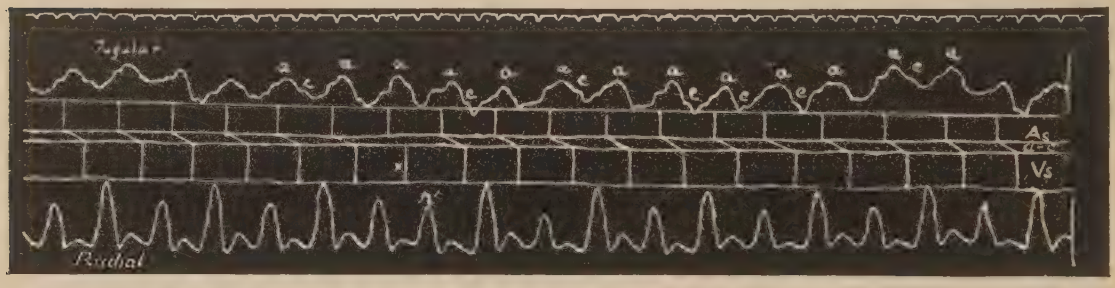


Fig. 221. Typical pulsus alternans with ventricular extra-systole r'. In the diagram it is shown to occur prematurely (downstroke x) and independently of the auricular stimulus. After the extra-systole there is a longer pause, and the alternating character of the pulse becomes more marked.

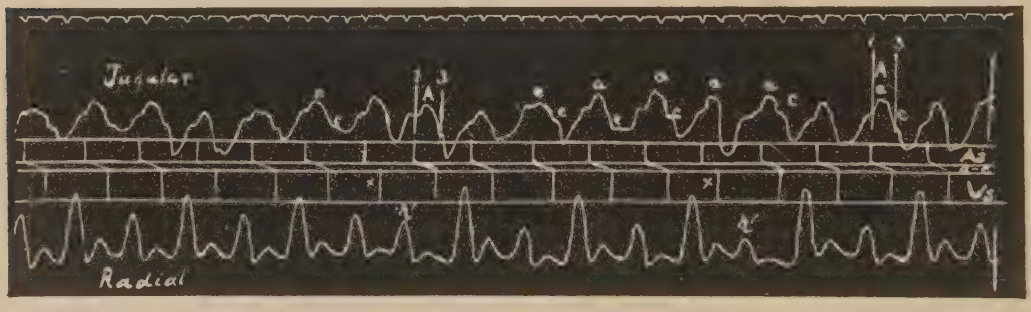


Fig. 222. Here there are two extra-systoles (r' r'), one after the small beat and the other after the large beat of the alternating rhythm.

tendency for pulsus alternans to occur in patients with a great increase of the pulse-rate due to an abnormal rhythm, especially in auricular flutter. There is not the same tendency with the normal rhythm, even when the heart's rate is increased from effort and other causes.

Clinical diagnosis.—It is only when the alternation is very marked that its presence can be detected by the finger, and this is rare. In rare instances it can be detected by a difference in the sounds.

When taking the blood-pressure its presence can frequently be made out, for the pressure of the big beats may be 10 or 15 millimetres Hg higher than the smaller beats. As the pressure in the bag is raised the finger on the pulse can perceive that the number of the pulse-beats is halved before the pulse disappears entirely. On listening at the bend of the elbow, the sounds in a like manner have become halved before they disappear as the pressure in the air bag is raised.

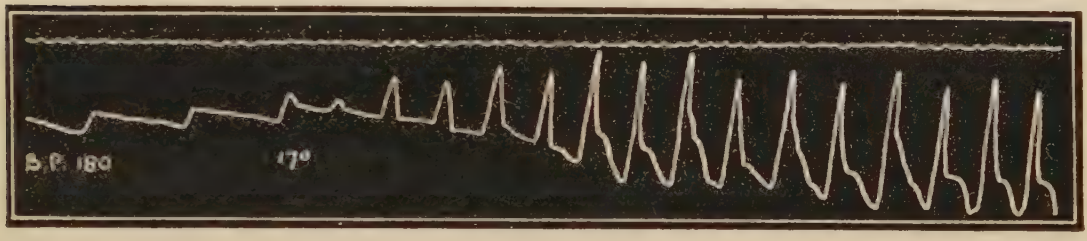


FIG. 223. Tracing taken from the radial artery after the air-bag connected with the manometer had obliterated the pulse at a pressure of 190 mm. Hg. The air was allowed to escape gradually, and when the pressure fell to 180 mm. Hg the sphygmograph recorded the stronger beats, and the smaller beats came through when the pressure fell to 170 mm. Hg. When the pressure in the air-bag was exhausted the pulse tracing showed rhythmical variation in the size of the waves—that is, the pulsus alternans.

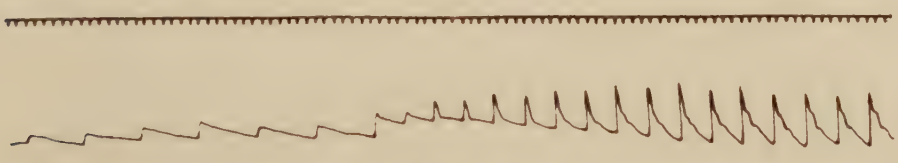


FIG. 224. Is a record of the pulse at the same time that the blood-pressure was taken. The air in the bag was raised till the pulse was obliterated. When the air escaped the pulse-beat returned. At first only every second beat escaped and moved the lever until the pressure fell further, when each beat was recorded. It is seen from this record that this method brings out the pulsus alternans when otherwise imperceptible or barely perceptible.

Figs. 223 and 224 show records of the pulse taken at the same time as the blood-pressure was being observed. After the pressure in the air-cuff had been raised till the pulse in the radial had disappeared, the air was allowed to escape, and as the pressure fell the pulse-beats came through and were recorded. It will be seen at first the rate was halved, but as the pressure fell all the beats were recorded.

The patient from whom Fig. 224 was taken had a pulsus alternans for three years, but it disappeared, and was not perceptible in a radial tracing as in the pulse tracing here, but when this record was taken it was shown still to be present.

Differential diagnosis.—There are some pulses which bear a surprising resemblance to the pulsus alternans. The one for which it is most commonly mistaken is the pulsus bigeminus, already described in Chapter XXIV. Here the smaller beat is due to an extra-systole and is always premature, and is followed by a prolonged pause. The difference is brought out by comparing Fig. 218 with 225. In Fig. 225 the interval between the big beat

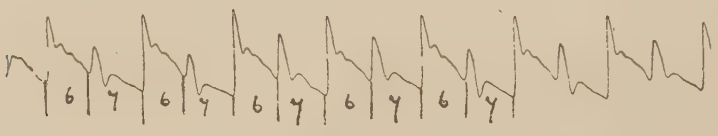


Fig. 225. Tracing of the pulsus bigeminus due to extra-systoles. The period between the big beat and the small is six-tenths of a second, and the period between the small beat and the big is seven-tenths of a second. (Compare with Fig. 218.)

and the small beat is $\frac{6}{10}$ of a second, while between the small beat and the big beat it is $\frac{7}{10}$ of a second. In the pulsus alternans, in Fig. 218, the interval between the large and the small beat is the same as that between the small and the large beat. Another somewhat rare form of resemblance is found in Fig. 226. This was taken from a healthy, vigorous man of 50, who complained of some dyspeptic symptoms. I had known for twenty years that he had a slow pulse, but I was surprised to see the apparent alternating character when taking a tracing. Listening to the heart, I detected, after each big beat, two short sharp sounds, characteristic of an extra-systole, and the explanation

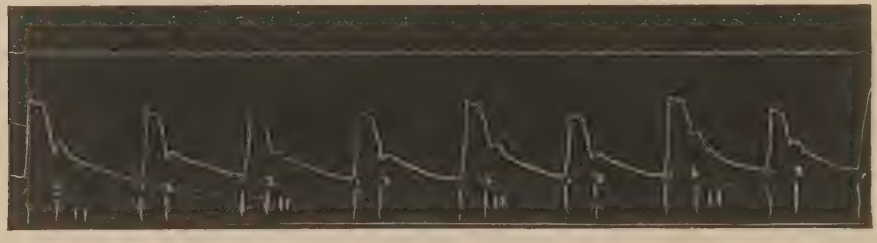


Fig. 226. Radial tracing simulating the pulsus alternans. On auscultation there were heard two short sharp sounds characteristic of extra-systoles after the large beat, but not after the small. These sounds are represented by the short lines in the tracing.

therefore was that an extra-systole occurred after every second normal beat, but of such weak force that no wave reached the radial. But the occurrence of the extra-systole had in some way caused a weakening of the following normal beat. Wardrop Griffith describes a somewhat similar experience.

Prognosis.—The prognostic significance in certain conditions of the pulsus alternans was recognized under the following circumstance. Many years ago in seeking for a differentiation of the different forms of pulse irregularity I

had placed in a group by themselves a number of tracings where the rhythm was regular, and where a big beat and a small beat occurred regularly after one another. Some of these tracings I published in a book on the pulse in 1902. In 1905 Wenckebach referred to these tracings, and also pointed out how they differed from other forms of irregularity. On reading his account I set out to re-examine my patients who had shown this condition, and found they were all dead. I then began to collect another series of records, and found that it was associated with a grave condition in certain patients, but that it had no such significance in other patients.

The conditions in which it is of grave significance are in those people in whom there are other evidences of cardiac impairment with the normal rhythm, this impairment being shown by breathlessness or pain on exertion. It is fairly frequent in patients with orthopnœa and cardiac asthma, but these signs in themselves are also of grave significance. The vast bulk of my patients have died within two years after the detection of its presence.

It is to be observed that it is only to be looked upon as a sign of gravity when associated with other evidences of marked cardiac impairment I have, on rare occasions, found it follow an extra-systole where it has had no grave significance.

OBSERVATION 62.—A man, 58 years of age, was brought to me by his doctor because of his irregular heart. I found an extra-systole frequently present, followed by a slight pulsus alternans. As the man was otherwise in good health, I told him to lead a normal life, attend to his business, and take as much exercise as he could without distress. This he did, playing golf and leading an active business life. This was in 1911. Three years later he again consulted me because, when on holiday, he had occasion to consult a doctor on account of a sore throat. The doctor warned him of the dangerous state of his heart because of the presence of a murmur and irregular action. On examination I found, in addition to the extra-systole, a systolic murmur at the base. I told him not to worry, but to carry on as usual, which he did. He called to see me in 1917 to be overhauled, and I found, in addition, an aortic diastolic murmur, but otherwise the heart was the same as before, and the response to effort good. He was advised to remember that he was getting on in years, and to live within the limits of his strength; and to-day, at the age of 71, he leads a quiet life, free from distress.

Pulsus alternans in such tachycardias as auricular flutter has no prognostic significance. It usually disappears when the normal rhythm returns, or becomes mixed up in the peculiar irregularity of auricular fibrillation when that condition replaces the flutter. The patient from whom Figs. 135 and 136 was obtained is still living, fourteen years after the record was taken (Observation 24).

CHAPTER XLIV

Combined Affections of the Genètic System

Auricular fibrillation and heart-block.
The effect of drugs.
The vagus effect upon the genetic system.

In reviewing all the Observations which have illustrated affections of this system, it can be seen that the suggestion put forward in Chapter XIII. is justified, in that all the disturbances of the rate and rhythm are due to the increased or decreased excitability or the cessation of activity of different parts of the genetic system. It is also seen that the peculiar variations are dependent upon the portion of the genetic system whose function is modified.

There are some other generalizations which support the views put forward here. So far, the disturbances of individual parts of the system have been dealt with. Such disturbances are not necessarily limited to individual parts, nor do the agents of disease act only on individual portions.

Auricular fibrillation and heart-block.—If we take up this last consideration we will find some instructive relations between agents which produce disturbances so wide apart, seemingly, as auricular fibrillation and heart-block. Both these conditions are more frequent in the later years of life, that is to say, the degenerative changes which are due to obliteration of the capillaries affect the structures of the s.-a. node and a.-v. node in the same way. The destruction of the s.-a. node and its connections in the auricle leaves the a.-v. node to start the contraction of the ventricle while the same changes may destroy the a.-v. node, leaving the bundle to start the contractions of the ventricle.

The disease agents which we associate with rheumatism are also liable to affect both the s.-a. node and the a.-v. node, for it is in people with rheumatic hearts that we find in the earlier stages of life auricular fibrillation and partial heart-block.

Indeed, if we search our records carefully we will sometimes discover a curious combination of auricular fibrillation and heart-block in the young who have suffered from rheumatic fever, and in the elderly.

Amongst the earliest Observations in which I employed the clinical polygraph to register the movements in the veins of the neck I found one which puzzled me greatly at the time, but which I now can see much more clearly. It was an account of a youth (Observation 63) who suffered from

a mild attack of rheumatic fever, who developed during his convalescence both partial heart-block and auricular fibrillation, as shown in Figs. 227-230.

A good many years ago I described several cases which I called ' nodal bradycardia '. These cases I can now recognize as being patients with

Fig. 227. Tracing of an irregular pulse due to partial heart-block from a patient convalescing from rheumatic fever. See Fig. 228. (Observation 63.)

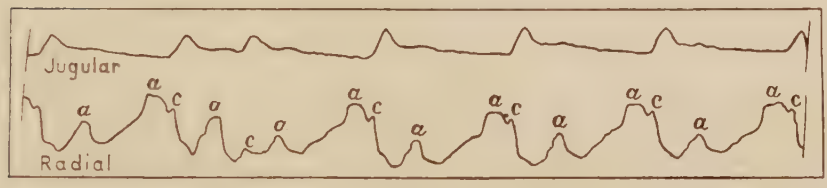


Fig. 228. Tracing of the radial and jugular pulses showing partial heart-block. (Observation 63.)

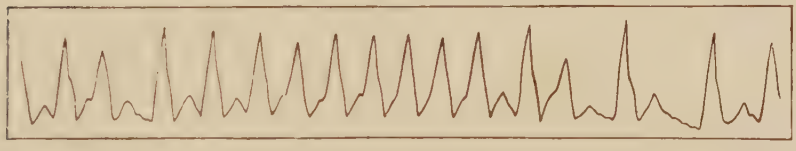


Fig. 229. Tracing showing an attack of auricular fibrillation. (Observation 63.)

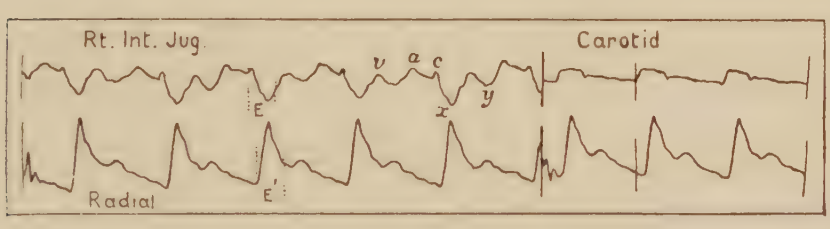


Fig. 230. Taken after complete recovery from rheumatic fever. (Observation 63.)

auricular fibrillation and complete heart-block. Two cases are cited in Observations 64 and 65. One was evidently due to senile causes, the other probably syphilitic in origin (Figs. 231-233). In Observation 69 both nodes were found degenerated.

In Observation 40 there is described a patient who suffered from influenza in which there occurred partial heart-block, that is, a disturbance

which affected the a.-v. node. But at one visit I found that the same disturbance had affected the genetic system in the auricle, so that impulses

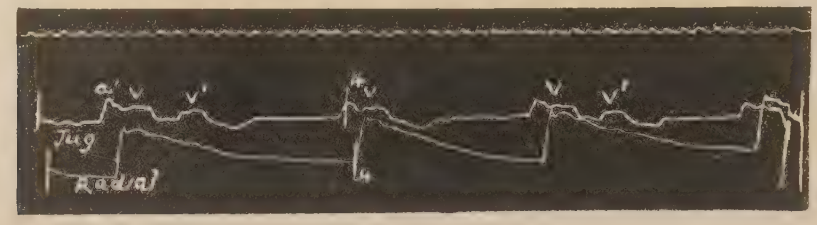


Fig. 231. Simultaneous tracings of the jugular and radial pulses. The jugular pulse is of the ventricular form. During the long pauses in the radial pulse there are small premature beats (v') in the jugular. The pulse-rate varied from 25 to 30 beats per minute. The patient was just recovering from a series of syncopal and epileptic attacks. (Observation 64.)

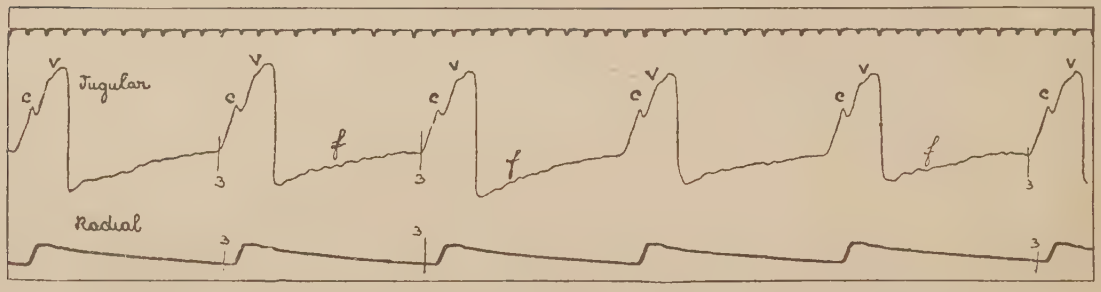


Fig. 232. Simultaneous tracings of the jugular and radial pulses. The jugular pulse is of the ventricular form, and the rate about 28 per minute. (Note auricular fibrillation waves f, f.) (Observation 65.)

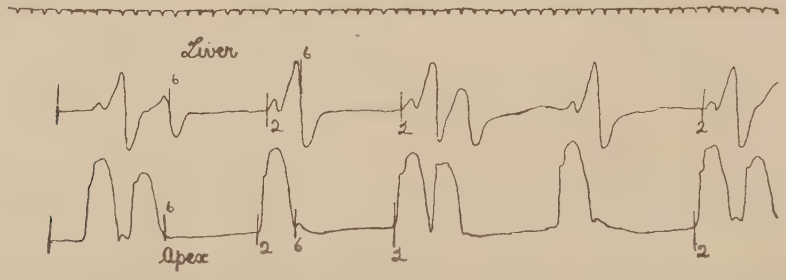


Fig. 233. Tracings of the liver pulse and apex beat in heart-block with auricular fibrillation showing the coupled beat. (Observation 65.)

from the s.-a. node were blocked, producing the condition called sino-auricular block (Fig. 234).

We occasionally meet some peculiar combinations of symptoms which it is not always easy to explain. In Observation 66, as the result of effort

there was a simultaneous onset of auricular fibrillation and complete heart-block which lasted only a short period and did not again occur (Fig. 235).

The effect of drugs.—A similar combination of symptoms may arise from the action of drugs. In one patient (Observation 67) there was advanced disease of the heart of rheumatic origin; digitalis would act at one time upon

FIG. 234. Shows the sino-auricular block. There is an absence of the auricular wave on two occasions in the spaces marked *o*. There is an absence also of the wave *x* in the radial beat. This patient had at the same time partial heart-block, as shown by the absence of the *c* wave and of a radial beat after some of the *a* waves. (Observation 40.)

the s.-a. node causing the heart to stand still sometimes as long as three seconds, at other times the rate was only decreased. A curious thing happened in the patient, in that although the *a-c* interval was increased when the rate slowed through the action of digitalis, the *a-c* interval decreased! Digitalis in rheumatic hearts with a wide *a-c* interval, or even when the interval is normal, frequently increases the interval and may cause a block so that beats drop out, but in this instance the digitalis actually caused a shortening of the interval. No doubt the longer period of rest which the a.-v. node obtained by the slowing of the rate, accounted for the shortening of the *a-c* interval, but the point is that the drug did not act upon the a.-v. node. More remarkable

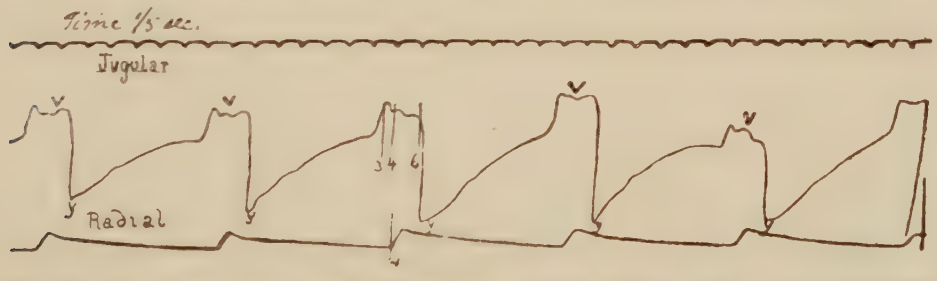


FIG. 235. Simultaneous tracings of the jugular and radial pulses. The jugular pulse is of the ventricular form. Pulse-rate, 40 per minute. (Observation 66, November 24, 1907.)

still was that digitalis in this case at other times caused auricular fibrillation—the effect here also being upon the s.-a. node, causing for the time being a cessation of its activity. When the digitalis was stopped the normal rhythm returned. The same result happened on another occasion. Later, this patient spontaneously developed auricular fibrillation, but the rate of the pulse was rapid (Fig. 239*a*), while when fibrillation developed under digitalis it was slow (Fig. 239). At another time digitalis produced extra-systoles (see Figs. 236-240).

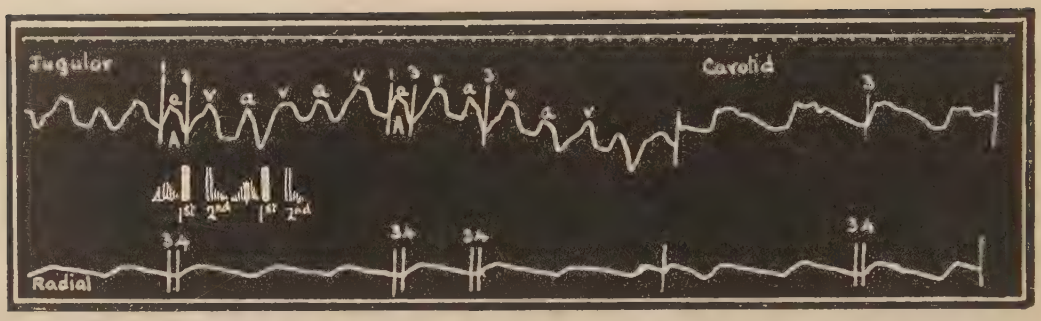


Fig. 236. Shows two pulsations in the jugular tracing (*a* and *v*) to one radial pulse. The *a-c* interval (space *A*) is increased. The shading shows the sounds of the heart and the murmurs present at the apex, viz. a presystolic murmur separated by a brief interval from the first sound, a reduplicated second sound followed by a diastolic murmur. These features of the jugular pulse and sounds and murmurs were always present when the patient was not under the influence of digitalis until the final establishment of auricular fibrillation. (Observation 67.)

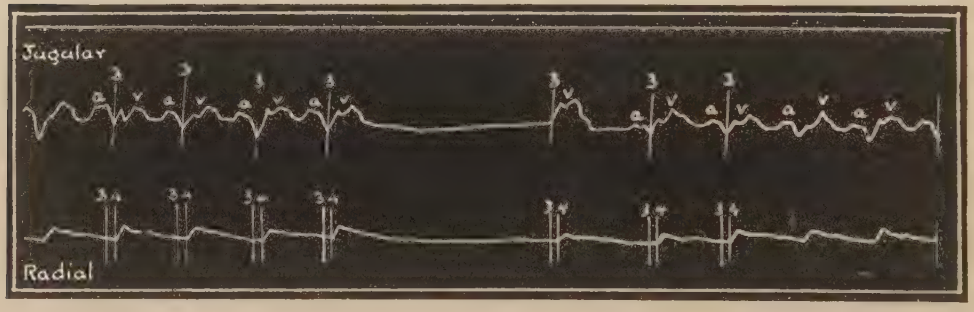


Fig. 237. Shows the temporary arrest of the whole heart from digitalis (Observation 67). The first beat after the long pause is an escaped ventricular beat, there being no auricular beat preceding it.

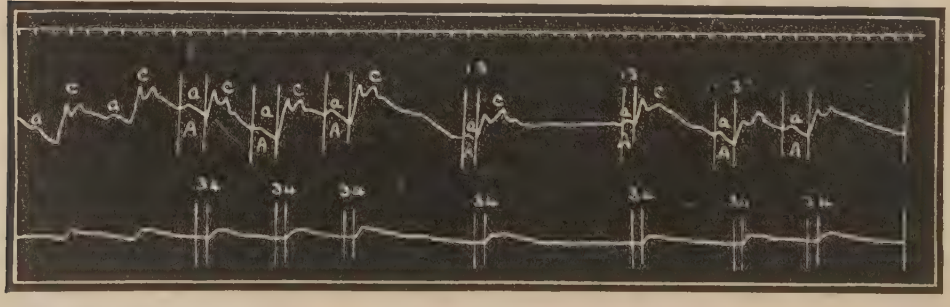


Fig. 238. The upper tracing was taken high up in the neck and shows a slight auricular wave (*a*) preceding the carotid pulse (*c*). After a long pause the *a-c* interval (space *A*) is much diminished. (Observation 67.)

It is very probable that no drug acts only on one organ. Its effect being mainly on the structures that regulate the organ's activity, the drug acts on structures of a like nature. Digitalis, in producing an effect on the heart, may cause at the same time vomiting and diarrhœa. Atropine produces

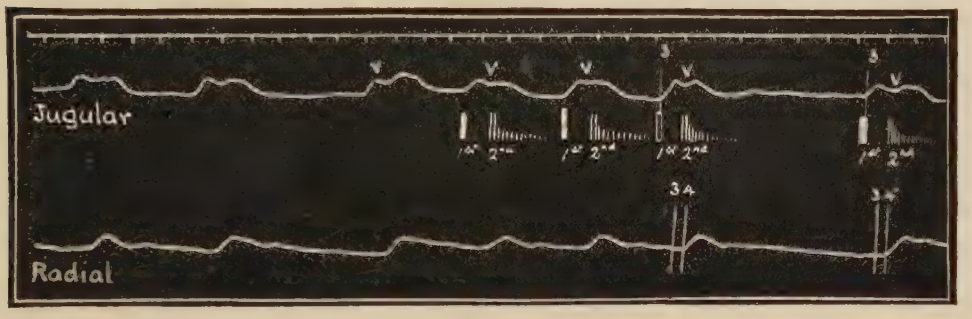


Fig. 239. Shows the inception of auricular fibrillation after digitalis. Compare the jugular pulse and the murmurs with Fig. 292. (Observation 67.)

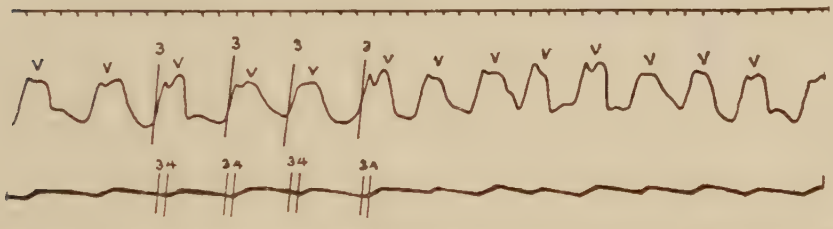


Fig. 239*a*. Shows the inception of auricular fibrillation without digitalis. (Observation 67.)

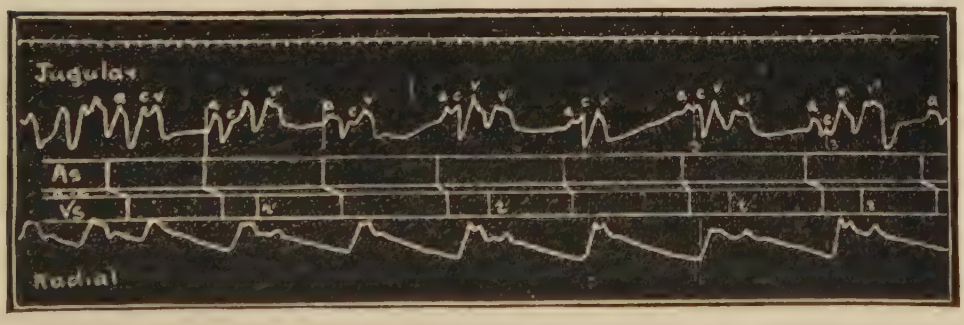


Fig. 240. Extra-systoles, *r'*, probably of ventricular origin, due to digitalis. The waves *v'* are due to the extra-systole. There are occasional long pauses when no extra-systole occurred, followed by a shortening of the *a-c* interval. (Observation 67.)

at the same time an increased heart-rate, dilatation of the pupil, delirium, dryness of throat. When a better knowledge is obtained of the structures and processes concerned in the functioning of organs, it will probably be found that the structures acted upon were all of the same kind.

Agents of disease have a tendency to affect structures of the same nature, and the disparity in the results or symptoms is simply due to the difference of the functions of the organs which are controlled by the structures affected.

Such disturbances due to disease agents modify the action of drugs, as one would expect when it is recognized that they both act on the same structure. It is possibly for this reason that digitalis gave rise to such varied and unusual results in the case just alluded to (Observation 67).

The vagus effect upon the genetic system.—In speaking of the effects of agents upon the genetic system I have dealt in somewhat general terms, describing the agent as one that depressed the excitability without specifying how this was brought about. It is conceivable that the agent acts directly upon the cells of the genetic system, reaching them through the blood stream, but the agent can also act through its effect upon the vagus and sympathetic nerves. It is not always clear which of these methods is concerned. It is well known that pinching of the frog's stomach will affect the heart through the vagus. The pressure on the vagus in the neck, or pressure upon the eyeball, also affects the heart, through the vagus acting either upon the s.-a. node or the a.-v. node, or upon both.

The question has often been discussed, 'Does digitalis act directly upon one or other of these nodes, or does it act through the vagus ?' That it does so by the latter method can be inferred from Observation 68. In this patient digitalis readily produced partial heart-block, and as the digitalis effect passed off some days after the drug was stopped, partial heart-block could be produced when the patient swallowed. Such a reaction could take place only through the stimulation of the vagus (Figs. 241-244).

It is to be observed that the act of swallowing as a rule causes an increase in the rate of the heart.

I am somewhat chary of dealing with this subject, for the facts are not yet sufficiently clear. It must be remembered that the conception of the heart-beat has hitherto been based upon insufficient data; the function of the genetic system and its influence upon the heart muscle have not been realized, so that the interpretation of the observations which have been made experimentally upon animal and on the human heart by the means of digitalis, atropine, and other drugs, all require reconsideration. In consequence, the discussions whether the drug acted upon the muscle or upon the nerve are now quite beside the mark. The whole matter has to be reviewed in the light of a better knowledge of the structures that are concerned in the heart-beat, and the part that each plays. It is for that reason that I chronicle in some detail clinical observations which throw light upon the subject, such as Observations 38, 67 and 68.

CHAPTER XLV

Cases illustrative of the Combined Affections of the Genetic System

Observations 63-68.

OBSERVATION 63.—Male. Aged 18. Examined February 7, 1892. His complaint was of weakness and a cough. The latter had persisted since an attack of measles in childhood. Beyond a slight pallor, nothing amiss could be detected in the examination.

In November 1892 he had a mild attack of rheumatic fever; the temperature did not rise above 102, and the pulse-rate was just under 100 per minute. There was nothing detected wrong with the heart. He was treated with salicylate of soda, and the fever gradually disappeared. On the third day after the subsidence of the fever, the pulse-rate fell to 60 beats per minute and became irregular, as in Figs. 227 and 228. A tracing taken at the same visit, of the jugular and radial pulses, shows that the irregularity of the rhythm and slowing of the rate were due to partial heart-block. The irregularity disappeared after a few days, but a fortnight later, when he called to see me, I found the heart's action was very rapid and irregular, as shown by the pulse tracing in Fig. 229. This condition lasted for several days—a week later the pulse was regular at the rate of 80 beats per minute—the jugular pulse showed a distinct lengthening of the *a-c* interval (Fig. 230). The patient had good health and was following his work.

I kept in contact with this patient for the next fifteen years, and he never showed any signs of cardiac impairment.

OBSERVATION 64.—Male. Born 1838. I had known this patient intimately since 1894. He was a healthy, vigorous man up till 1907. He was a very heavy smoker, and for a great many years he smoked two ounces of tobacco and half-a-dozen cigars a day. I had occasion to examine him in 1906, and found his heart normal in rate and rhythm, though for some years he had been rather short of breath. I again examined him in February 1907, and found that his heart was continuously irregular, with the disorderly rhythm characteristic of the auricular fibrillation. He was not conscious of the change, but there was a further increase in his breathlessness. He was still able to attend to business, and to play a game of golf. He lived some distance from me, and I did not see him again until October 11, 1907, when I was asked to see him with his medical attendant, Dr. O'Connor, to whom I am indebted for an excellent account of his many seizures. The history given was that his pulse had become very slow for some months, and that latterly he had been seized at times with attacks of unconsciousness. The pulse-rate on such occasions was found below 30 beats per minute. He was very weak and faint when I saw him, the pulse varying in rate from 30 to 40 beats per minute, and irregular, with long pauses at times. During the long pauses there was often a small premature beat in the jugular (see *v'*, Fig. 231). The heart's dullness extended 1½ inches beyond the left nipple, and there was a soft blowing murmur at the apex. There was a small amount of albumin in the urine. The attacks of unconsciousness continued, and I saw him again in November, when the heart's condition was much the same. After this the pulse-rate increased, the attacks disappeared, and he went to Torquay in June 1908, where he had a slight recurrence of his attacks of loss of consciousness. From this he recovered, and continued well till August 4,

when after some effort he was seized with great breathlessness, and the attacks of unconsciousness recurred. These attacks increased in number and severity, and once for two whole days he was unconscious and deeply cyanosed. For some hours he passed from one epileptic seizure to another. He also developed Cheyne-Stokes respiration. The pulse during these convulsive attacks was not perceptible. The severity of the attacks gradually lessened, and in the month of September his pulse-rate rose to 50 or 60 beats per minute. In October he had a number of very transient fainting attacks, and his doctor described the attacks as resembling *petit mal*. Thus, while the doctor was talking to him, the patient's face would suddenly become pale, and consciousness would be lost for a brief period. During these attacks no pulse could be felt at the radial.

I saw him again on December 18, 1908. He was able to go about, and had been free from attacks for a few weeks. The pulse was rather slow, about 60 per minute, and irregular (auricular fibrillation). The heart's dullness extended 1½ inches beyond the left nipple, the sounds were clear, with a faint doubling of the first sound. There was no dropsy, and the urine was free from albumin.

A record taken on May 5, 1909, showed a rhythm of a disorderly kind, characteristic of auricular fibrillation, and the jugular pulse was of the ventricular form. In August 1909 the heart's action became very infrequent, and he was seized with attacks of loss of consciousness and epileptic convulsions, and he died in one of these attacks.

The heart was examined by Dr. A. E. Cohn, who reported increase of the connective tissue of the sino-auricular node. There were also present interstitial myocardial lesions of the auricles and ventricle, but more especially of the right auricle, but there was only slight increase of connective tissue scattered through the auriculo-ventricular node and branches.

OBSERVATION 65.—Male. Born 1865. When a soldier in India he had dysentery at the age of 20, syphilis at the age of 22. He had malarial fever in America at the age of 27. In 1894 he had the first attack of syncope. After lying up for a week he went out, and in hurrying to avoid a cab he fell unconscious on the pavement, but quickly recovered. He consulted a doctor, who said his heart was affected. Two years later he was laid up with shortness of breath and swelling of the legs, and was treated for 'mitral disease'. He partially recovered, and had frequent attacks of weakness until the final breakdown occurred in 1905. He had been feeling ill for some years, but had worked hard, and had kept himself going on brandy, bovril, eggs, etc. He says his pulse was slow in 1903, and that it has remained so ever since.

In 1904 he began to have mild 'fits', in which he lost consciousness and was slightly convulsed. From November 1905 to April 1906 he had a great number of fits, some severe with convulsions and cyanosis, others slight without convulsions. He had no attacks for a year, but he had a very severe one in April 1907, and since then only three mild attacks. He had lived a life of hard work with frequent bouts of drinking.

Examined in 1910, the patient was tall, spare, and intelligent-looking. The face was usually ruddy, with a faint duskiness. He walked slowly and carefully, and his gait was slightly ataxic; if he hurried or got excited he became giddy. He had a somewhat violent temper, and when in a passion his face became dusky and cyanosed. When lying down there was a large pulsation, seen in the deep jugular veins on both sides, heaving in the lower part of the neck, as in Fig. 232. It was very slow and synchronous with the apex beat. The liver was slightly enlarged and pulsated (Fig. 233).

The radial pulse was slow and deliberate, usually about 30 per minute, and quite regular. At times two beats were close together, and were followed by a long pause. These coupled beats might appear at rare intervals or alternate with a single beat, or they might appear continuously for a short period. The apex beat was large and diffuse in the sixth interspace, and in the anterior axillary line. The heart dullness extended 1 inch to the right of the middle line and 8 inches to the left.

There was a rough blowing systolic murmur, heard best at the apex and propagated towards the axilla. The second sound was clear and well struck and followed by a soft murmur. This diastolic murmur was heard only over a limited space at the apex, and was not always perceptible. It was, as a rule, faint and faded away.

A large number of tracings were taken from this patient at different times, and they always presented the same features, the only difference being that sometimes the coupled beats were more frequent or were entirely absent. The jugular tracing always showed one large wave occupying the whole time of ventricular systole, occasionally with fibrillation waves during diastole.

On July 6, 1911, he began to have a series of attacks of loss of consciousness and slight convulsions, which occurred at varying intervals till his death on July 8. The heart at times varied slightly in rate and rhythm. In place of the usual slow rhythm, a series of beats, probably of the same nature and origin as the extra-systoles, would follow one another for varying periods, so that the ventricular rate sometimes attained a speed of 60 per minute. At the end of such a period the ventricle would stand still before it again took on its own rhythm, and it was during this period that the loss of consciousness and slight convulsions occurred. The attacks of unconsciousness and the mild convulsions commenced at 5 o'clock on the morning of July 6, 1911. They ceased at 9 A.M., but returned at 11 A.M., and continued at short intervals till 1 P.M. Between 1 P.M. and 2.30 P.M. he was free from them. From this hour until death occurred (the morning of the 8th) there was a similar repetition of relapse and recovery. I am indebted to Dr. Silberberg for the following description.

The attacks were of varying duration, lasting for a few seconds to twenty or thirty seconds. They were accompanied, one and all, by a lapse of the ventricular beats, readily observed at the prominent apex beat. The onset of unconsciousness was gradual, and the patient was aware of the impending attack, being conscious that his heart had temporarily ceased to beat. He became restless, he groaned, and uttered words of complaint. In a few seconds he could not be roused ; the eyes rolled upwards and deviated to the right, the pupils dilated fully, and the corneal reflex was lost. Cyanosis of the face, already present, deepened to lividity ; the breathing became stertorous and air was sucked in vigorously, his cheeks sinking deeply between his edentulous gums. Much flatus was passed. After twenty seconds of unconsciousness, epileptiform manifestations appeared ; the convulsions started in the face, the arms became rigid, and spasmodic flexor movements appeared. The lower limbs showed no convulsive movements. A single beat of the heart, during the fit, lightened the degree of unconsciousness, two or three beats brought him to a dazed condition, and after a few more beats he rapidly recovered, conversing rationally, though necessarily showing exhaustion. The first beat of the recovery was usually a weak one, the ensuing beats were more forcible. During the periods of comparative lucidity he complained of aching and sore feelings all over, more especially in the limbs. He was too feeble and exhausted to move. Pain, relieved by eructation, was present over the upper abdomen. On several occasions he vomited a pint or more of greenish fluid, an incident which seemed to afford relief.

Dr. A. E. Cohn examined the body and the following is a summary of his report :

' The medulla oblongata showed no gross lesion. The vagus nerves were normal, but parallel with each nerve there was a small one containing a hæmorrhage. The heart was hypertrophied in all its cavities. There was an aneurysm in the right upper portion of the left ventricle. There was partial sclerosis of the septum ventriculorum and complete sclerosis of the septum membranaceum. The myocardium contained numerous scars. There was practically no acute inflammation. The sino-auricular node was in part destroyed, being replaced by connective tissue. The main stem of the auriculo-ventricular bundle was divided from the auriculo-ventricular node by sclerotic tissue ; and the distal end of the main stem, its point of division, and the upper parts of both branches were destroyed by the same process. The arteries of the heart showed hypertrophy of the media, degeneration of their muscle fibres, and hyperplasia of the intima, causing either partial (the more common lesion) or complete obliteration of the lumina. The aorta showed athero-sclerosis. The liver showed chronic congestion, as did also the spleen, pancreas, and kidneys.'

OBSERVATION 66.—Male. Born 1852. A stout, healthy-looking individual. I had known him for about twenty-eight years, and had attended him at various times for trivial complaints, and in 1903 for an attack of erysipelas of the face. He had enjoyed good health, was getting fat and somewhat short of breath. On November 9, 1907, he was hurrying from

the train to a football match a mile distant from the station. As he approached the football field he was seized with pain across the middle of the chest, but as it was not severe he pushed on till he arrived at the field. He sat down, but the pain increased, striking into both arms, and his hands went white and cold. He felt as if he wanted to breathe deeply but could not. He endured the suffering for twenty minutes, and as it became worse, and he felt as if he would die, he was assisted off the field, put into a cab, and driven to the station. He was given some brandy, which made him sick. The pain gradually diminished, and he returned home by train; as he was better he walked home (about a quarter of a mile), but felt sick and short of breath. He went to bed, and one of my colleagues saw him and found his pulse between 30 and 40 beats per minute. I saw him next morning. He felt very weak; the pain was nearly gone, though it had kept recurring through the night. He had some pain if he took a deep breath. The pulse-rate was 52; the heart's dullness extended from mid-sternum to 2 inches beyond the nipple line; apex beat faint in the fifth interspace; sounds clear and free from murmur. The superficial jugular vein was very full, but did not pulsate. The deep jugular was large, filling up during ventricular systole, and collapsing suddenly at the beginning of ventricular diastole. There was no sign of an auricular wave preceding the ventricular systole. The patient was kept in bed, and his condition did not undergo much change for the next fortnight, except that the pain gradually grew less till it finally disappeared and he was able to sit up. The pulse-rate varied, sometimes falling as low as 30, but never rising above 52. On November 24 a long tracing was taken with the ink polygraph, and the rate showed great uniformity; the rhythm was also quite regular. Fig. 235 is a small portion of the tracing taken on that day, and represents the same features as were present on November 10; the rate was 40 per minute, the rhythm regular, and the venous pulse of the ventricular form. When I next examined him, on November 29, his pulse had increased in rate, with occasional intermissions. I had the greatest difficulty in getting a tracing of the jugular pulse; he had a very short fat neck. But imperfect as the tracings were, they showed a return of the auricular wave *a* to its normal period before *c*. From this date he gradually improved, and has been able to get about, though he is perhaps a little shorter of breath than before his attack. Tracings were taken from him in December 1908, and on May 11, 1909. The venous pulse was the same on both occasions, and showed an auricular wave, *a*, preceding the carotid wave, *c*, while the rate was 68 per minute; the rhythm was regular. In 1924 he was in fair health and had no return of the attacks.

OBSERVATION 67.—Female. Aged 28. Consulted me on April 24, 1907, complaining of swelling of the abdomen, shortness of breath, and palpitation. She began to feel ill in November of the previous year; she had rheumatic fever fifteen years previously. The face was dusky, the breathing laboured, the legs and abdomen swollen. She passed a small amount of urine. There was rapid pulsation in the veins of the neck—two to each radial pulse (Fig. 236)—the pulse was small and regular, blood-pressure 100 mm. Hg. A thrill systolic in time could be felt over the upper part of the chest. The movement of the heart farthest to the left was 3 inches beyond the nipple, and showed an indrawing during ventricular systole (giving rise to an inverted cardiogram). The heart's dullness extended half an inch to the right of the sternum. At the apex there was heard a presystolic murmur, a diastolic murmur, and a reduplicated second sound. At the base there was a loud, rough murmur systolic in time, heard also over the carotid. There was also a slight diastolic murmur at the aortic area. Over the heart's dullness inside the nipple there was another murmur systolic in time, and while listening one could imagine that two hearts were working, a series of sounds being heard immediately under the stethoscope, while another series of sounds could be heard faintly and seemingly at a distance.

Sometimes when the heart was regular but slow, a confusing change took place. During the diastole of the heart there was a sudden increase in the intensity of the diastolic murmur, in fact a mid-diastolic murmur followed after a pause by the first sound. When I drew the position of the murmur under a tracing I found it corresponded to the position of the auricular wave in the jugular tracing (Fig. 236), and no doubt it was due to the auricular systole. When the heart became very slow, because of the digitalis, the longer rest gave the nodal cells time to recover and the murmur was heard nearer and nearer the first sound, and sometimes it

ceased to be heard. This only happened when the heart became very slow and the auricular wave in the jugular approached quite close to the carotid, as in Fig. 238. The patient was put on the digitalis, squills, and calomel pill, one taken three times a day. On May 1, after taking eighteen pills, she was passing more urine. She felt much better on the 8th—the swelling had gone from the abdomen and the legs were less in size. On the 15th the swelling had all gone, she had a little diarrhœa and felt sickly, but breathed easier and was able to walk better. The heart was slow and irregular. On examination there was only one wave visible in the jugular, and when recorded it was found synchronous with the carotid pulse (Fig. 239). There was no presystolic murmur, but only a long diastolic murmur at the apex. The tracing shows the jugular pulse to be of the ventricular type; *auricular fibrillation had occurred.* The pills were stopped, but the heart was still irregular on the 19th. On the 26th it was regular and the jugular pulse double waved, as in Fig. 236. She felt better, but the legs and abdomen were swelling again. Digitalin granules were prescribed, one three times a day. On the 31st the pulse became irregular, and tracings taken on this day and on June 2 showed that it was due to the slowing of the whole heart. On June 4 the heart had again taken on auricular fibrillation, which persisted until the 17th, when the auricular contractions appeared and the heart showed frequent pauses. She was taking one granule of digitalin per day until the 23rd, when it was

Fig. 241. The jugular tracing shows a wide *a-c* interval (space *A*).
(Observation 68, before digitalis.)

stopped, and on the 28th the heart was found rapid and regular and the pulsation in the veins double waved, as in Fig. 236. Up till November 4 these reactions due to digitalis continued to appear; sometimes auricular fibrillation would appear and sometimes the long pauses as in Fig. 237, and occasionally extra-systoles (Fig. 240). A few days after stopping the digitalin the heart invariably became regular. On November 6, after digitalin treatment had ceased, auricular fibrillation started spontaneously, the heart beating rapidly, but it could be slowed by digitalis or strophanthus.

Records of blood-pressure showed generally a fall to 100 mm. Hg, when there was much dropsy, and a rise with slowing of the heart to 135 or 140 mm. Hg. Sometimes, however, the pressure was 130 with dropsy, and no increase occurred with its disappearance and the coincident improvement of the patient's condition after taking digitalis.

The patient died in March 1908.

OBSERVATION 68.—Male. Aged 25. Consulted me for a stiffness and swelling in sundry joints, wrists, ankle, and knee, on May 4, 1906. The heart was rapid in its action, 120 per minute; slightly enlarged, with systolic mitral and tricuspid murmurs. There was marked pulsation in the neck, of which Fig. 241 is a tracing. The movement due to the carotid was always large, and forms a marked feature (wave *c* in all the tracings). In the course of the next fortnight there gradually developed double aortic murmurs. By May 23, under treatment, he had gradually improved, the rate of the heart falling to 90 beats per minute.

On May 24, one granule of Nativelle's digitalin granules, $\frac{1}{200}$ of a grain, three times a day, was prescribed.

I kept him under observation, but could detect no change in the heart's action until May 30, after he had taken nineteen granules. On this date I found the pulse at times irregular. Fig. 242 is a tracing, showing the nature of the irregularity. Between the jugular and radial pulses I have intercalated a diagram illustrating the events in the tracing from the neck. It will be noticed that before a ventricular beat drops out there is a gradual lengthening of the *a-c* interval,

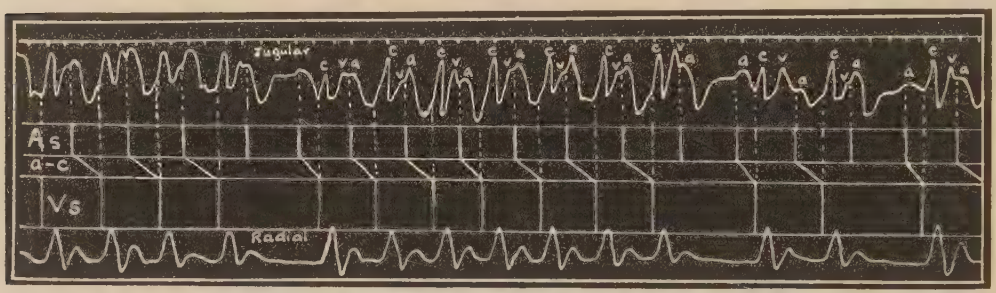


Fig. 242. After taking nineteen digitalin granules the pulse became irregular, which the intercalated diagram shows to be due to dropping out of the ventricular systoles—a mild form of heart-block. (Observation 68.)

and that the dropping out of the ventricular systole is manifestly due to an increased depression of the conductivity of the fibres joining *a* and *v*—that is to say, the stimulus from the auricle is blocked before it reaches the ventricle. The digitalin was stopped, and a few days later all signs of irregularity had disappeared. The patient himself was conscious when his heart was irregular, and I remarked to him that the irregularity had gone; he replied, 'I can bring it back'. I asked him how he could do so, and he said, 'By swallowing'. I asked him to swallow, and he did so, and immediately I detected long pauses in his pulse, while, on auscultation of the heart, no sounds were heard during the pauses. I took a large number of tracings for an hour and a half, during which time he swallowed forty or fifty times, and the alteration in the pulse-rate never failed to appear. The characteristic changes are seen in Figs. 243 and 244. After

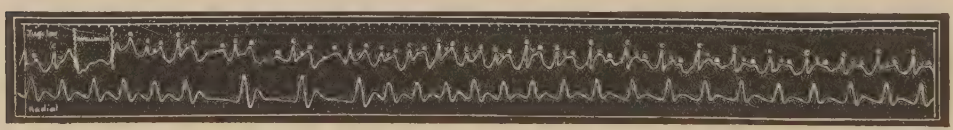


Fig. 243. Shows a reflex effect of the vagus due to swallowing. After the act of swallowing the pulse became very slow, because of the dropping out of the ventricular systoles (see diagram in Fig. 244). After this the heart's rate increased slightly, then became slow again. (Observation 68.)

swallowing there are each time three regular beats, then the pulse slows in the manner shown in the tracings. After two or three slow beats the rate of the heart increased for six or seven beats, then gradually slowed in the manner shown in the latter part of the tracings. Occasionally during the secondary slowing one ventricular systole would drop out, as is shown in Fig. 244. In Fig. 244 there is intercalated a diagram which shows the nature of the arrhythmia, and it can there be seen that the long pauses in these tracings are preceded by an increase of the *a-c* interval, just as happened when the patient was under the influence of digitalis (Fig. 242), and that the dropping of the ventricular systole was due to a block of the stimulus from auricle to

ventricle. The numbers given under the radial tracings in Fig. 244 represent tenths of seconds, and from these numbers the manner in which the rate of the pulse varies can better be realized.

The susceptibility of the heart to the act of swallowing continued for a week, then entirely disappeared. There is no doubt it arose through reflex stimulation of the vagus induced by

FIG. 244. Shows the same as Fig. 243, except that during the second period of slowing after swallowing there is a long pause due to the dropping out of a ventricular systole.

the act of swallowing. The analogy between the effects of digitalis and of reflex inhibition induced by swallowing appears worthy of note, as it indicates that the action of the drug here is exerted through its effects on the vagus centre, and not through the changes it induces in the heart-muscle directly.

CHAPTER XLVI

The Genetic System in the Production of Death

The suspension of the activity of the conducting system.
Ventricular fibrillation.

In the description, so far, of the various disturbances of the genetic system only two are shown to lead directly to death. These were the stimulation of the ventricle to rapid and imperfect contractions and the standstill of the ventricle, so that the output of blood was stopped or became so slight that death followed. These occurred in auricular fibrillation and flutter, and in complete heart-block. Death may arise in two other ways—from the failure of the genetic system to function and by ventricular fibrillation.

The suspension of the activity of the conducting system.—It has been long recognized that with the cessation of respiration the heart gradually ceases to beat. The manner in which this is brought about is chiefly by a cessation of the activity of the a.-v. node and bundle. In hibernating animals like the bat, as Miss Buchanan has pointed out, the heart becomes slow and intermittent due to partial heart-block. In asphyxiated animals the same thing happens. In the dying heart certain portions of the auricle—the 'ultimum moriens'—continue to beat when the ventricle does not respond. It is evident from this that though the stimulus for contraction can arise either in the s.-a. node or some part of the genetic system in the auricle, impulses are not conveyed to the ventricle, which stands still, and in consequence the cessation of output leads to death.

Ventricular fibrillation.—MacWilliam[1] pointed out in 1889 that a common cause, perhaps the most common cause, of sudden death was due to the ventricle passing into fibrillation. He attributed this condition, as also the fibrillation of the auricle, to the sudden shortening of the refractory period of the muscle fibres. On the other hand, I favour the view that the fibrillation is due to the loss of control of the regulating structures of the heart, for the reasons given in discussing the problem of auricular fibrillation.

It has already been pointed out how the a.-v. bundle differs from a nerve, in that when a nerve fibre is cut off from its receptor and its cell body, it fails to function. The bundle, on the other hand, is made up of a great series of

[1] *Brit. Med. Journ.*, 1923, ii. pp. 215, 278.

cells, so that when it is cut and separated from the a.-v. node, the cells continue to function, and in doing so exercise a control over the muscle.

The junctions between cells of different kinds are the places most susceptible to injurious influences. This general proposition would indicate that the place where disturbances are apt to occur which would produce ventricular fibrillation is at the junction between the bundle cells and the muscle cells. Such a disturbance leads to the cessation of impulses from the bundle, then the ventricle passes into fibrillation.

In repeating this explanation, I do not insist that it is correct, but it is one which seems to bring this condition into relation with others of a like kind, where the organ gives evidence of disorderly and purposeless activity.

This purposeless activity results in the complete cessation of the output of blood from the ventricle, and death is thereby induced.

A number of observers have taken electro-cardiograms from dying patients, and have demonstrated that death sometimes ensues with the passing of the ventricle into fibrillation.

The only occasion in which I observed ventricular fibrillation is described in Observation 60, p. 282. I did not recognize the nature of the pulse tracing, but Professor MacWilliam told me it was typical of attacks of ventricular fibrillation. Between the attacks the patient showed no sign of impending death, and it is very probable that in this instance the death was not due to standstill of the ventricle, as is usual in heart-block, but to ventricular fibrillation.

CHAPTER XLVII

Affections of the Myocardium

Nature of the symptoms.
Increased size of the heart.
Dilatation of the heart.
Effort and dilatation.
Dilatation and heart failure
Muscle failure.
Prognosis.
Treatment.

Nature of the symptoms.—So far, the subject of heart affections has been described mainly from the point of view of that part of the reflex arc concerned in the production and conduction of impulses. The next part of the arc to be considered is the effector organ, *i.e.* the muscle fibres. It will be convenient to discuss the symptoms produced by the muscle fibres in three ways: First, the alteration in the size and shape of the heart; second, the results of an impaired output; and third, the reflex symptoms.

Before setting out to deal with the matter under these three headings, it would be well to point out the reasons for this division—they are chiefly clinical. An attempt to describe the subject from the nature of the disease of the muscle would fail, for there are no signs distinctive of any one disease. If the function of the muscle is not impaired, the disease does not affect the health of the individual, but if the function is impaired, then the symptoms become definite, but they are common to all forms of disease, being due either to a defective output, as shown by breathlessness and dropsy, or to the involvement of the nervous system, as shown by pain and other signs of angina pectoris.

Increased size of the heart.—An increase in the size of the heart may be due to three conditions: (1) muscular hypertrophy; (2) increase in the thickness of the walls by increase of fibrous tissue, or other abnormal structure, such as gummata; and (3) dilatation of the heart. It is seldom possible to tell, with certainty, which of these conditions are present. In aortic regurgitation, and in adherent pericardium, we may justly assume that the increase in size is due to muscular hypertrophy, but when the increase of size is due to diseased changes in the heart wall there is no feature on physical examination to distinguish it from either hypertrophy or dilatation. We may

conclude, from a large and forcible apex beat with an outward thrust during systole (Fig. 245), that the left ventricle is increased in size, but that is not necessarily evidence of hypertrophy. A diffuse apex beat with an in-drawing of the impulse during systole and the out-thrust during diastole may be recognized (Fig. 246) as evidences of an increased size of the right ventricle, but it does not tell whether it is dilatation or hypertrophy. An

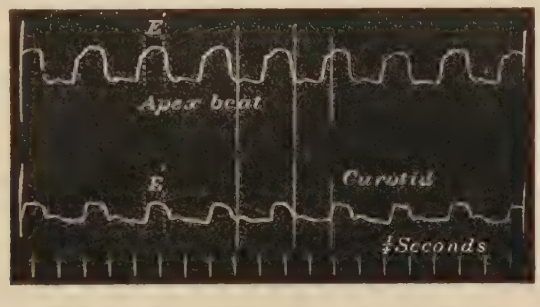


Fig. 245. Simultaneous tracings of the apex beat and the carotid pulse, showing the 'systolic plateau' in the cardiogram during the outflow from the ventricle (E'). The out-thrust of the apex is due to the left ventricle.

increase in the extension of the dullness beyond the right edge of the sternum we may recognize as an evidence of the dilatation of the right auricle.

The displacement of the heart that takes place in dilatation of the right auricle and ventricle is seen in Figs. 247 and 248. It will be seen in these figures that increase in the size of the right side causes the heart to extend

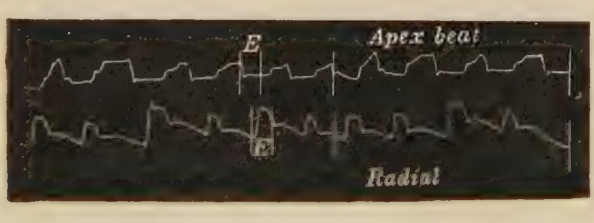


Fig. 246. Simultaneous tracings of the apex beat, and of the radial pulse. The rise in the apex tracing resembles the usual characters present in a tracing of the apex beat due to the left ventricle. On analysis, it is found that the elevation is during the diastole, and the fall (E) during the systole, of the ventricle, and therefore the apex out-thrust is due to the right ventricle.

more to the left than the right, the reason being that the pericardium, which extends from the aorta and pulmonary artery and veins above to the inferior vena cava below, prevents its extension to the right and forces it to the left.

While an examination by the X-rays shows an increase in size, it gives no information of the part enlarged or the cause of the enlargement.

The information derived from the size of the heart is thus limited. We can surmise as to the nature of the change, but such surmise does not afford

us the kind of knowledge wanted. That is to be found by estimating its functional efficiency, and this can only be done by finding out how the circulation is carried on when the body is at rest and on the performance of certain forms of effort.

Dilatation of the heart.—Although the size of the heart yields little information of the kind wanted, it is necessary to understand something of that still obscure condition—dilatation. Dilatation is so connected with the 'tone' of the heart that it is impossible to understand the one without the other.

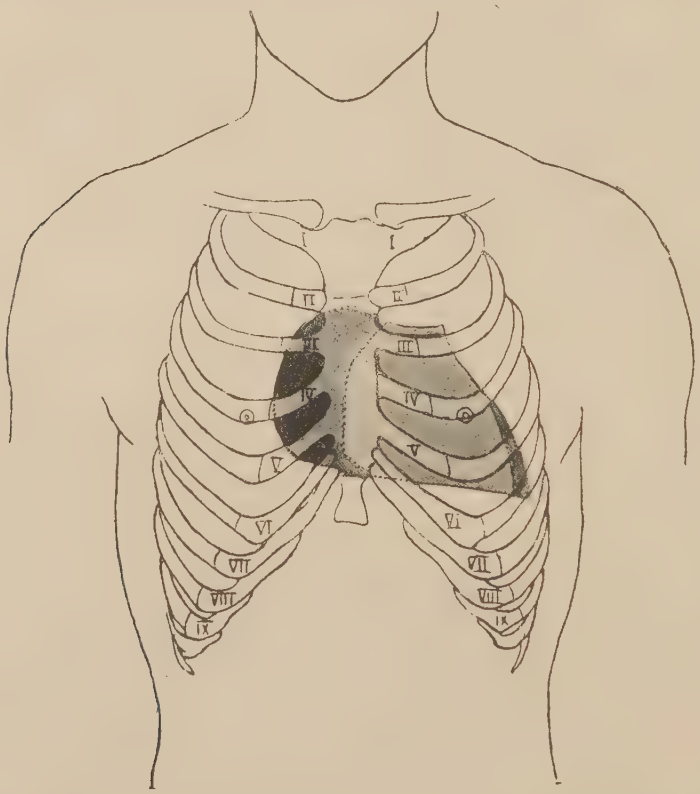


Fig. 247. Position of the chambers of the heart in extreme dilatation, as in the late stage of mitral stenosis with auricular fibrillation. The portion of the heart on the right of the sternum shaded deeply, together with that behind the sternum up to the dotted line, represents the right auricle, while the small strip to the left shaded deeply represents the left ventricle. The part between is the right ventricle. (Harris.)

Some say there is no such thing as tone of the heart muscle, that dilatation is produced only by the force exercised by the blood upon the walls of the heart. But if it be asked, whence comes this distending force, the inadequacy of such an explanation is at once apparent. During systole, the increased pressure within the chamber is produced by the contraction of the wall of the chamber itself, and one can scarcely assume that in the process of contraction

dilatation is produced. Dilatation of an auricle, it is true, might be produced by the forcible regurgitation of blood from a powerful ventricle, but such a thing can only happen when there is a lesion of the auriculo-ventricular valves. Regurgitation, apart from valvular lesion, can only occur after the muscle fibres surrounding the auriculo-ventricular orifices have become relaxed—that is to say, dilatation of the auricle from such a cause would be produced after dilatation of the ventricle.

That neither the resistance opposed to a chamber during the systole nor

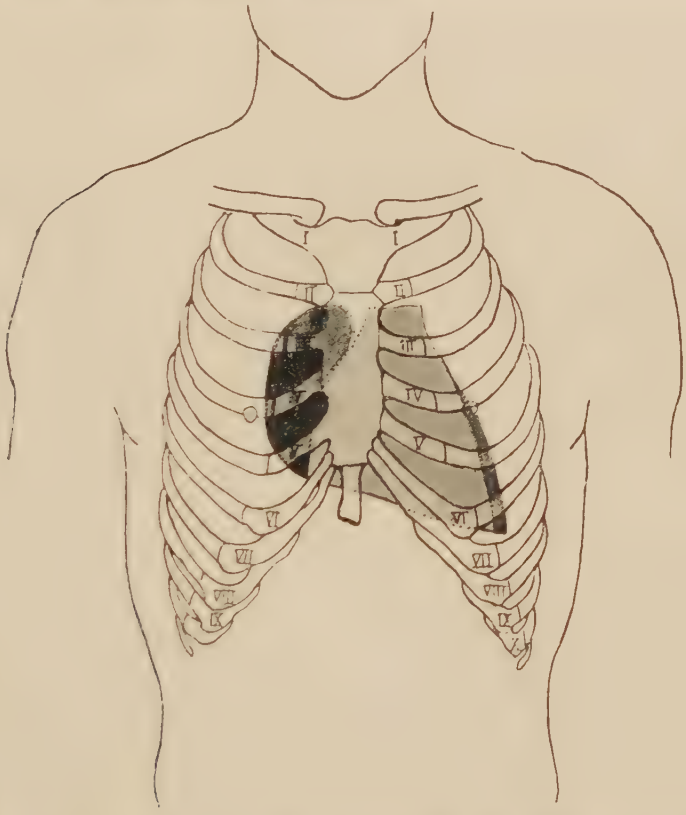


Fig. 248. Dilatation of the heart with the apex beat displaced downwards. The shading is the same as in the preceding figure. (Harris.)

the distending force during its diastole is the cause of dilatation becomes evident when the conditions observed in certain hearts are carefully studied. Thus hearts whose walls are thinned, and whose muscle fibres are degenerated, may continue to work against an abnormally high arterial pressure, and never show any signs of dilatation. In fact, the wall of the left ventricle may be so thinned that it actually bursts in its effort to overcome the aortic pressure, yet the walls show no signs of dilatation. Sir Arthur Keith, who has especially looked into this matter of ruptured hearts, informs me that such hearts may

show no sign of increase in the size of their cavities. In a patient under my care, the heart was so enfeebled that he could scarcely walk fifty yards without inducing an attack of angina pectoris, but no enlargement of the heart could be detected. He died suddenly from rupture of the heart, and I found that part of the heart-wall was so thinned as to be made up of little except the endocardium and pericardium, yet, notwithstanding this enfeeblement of the heart-wall, there was no sign of dilatation of the cavity.

Dilatation of the left ventricle may occur even when the diastolic force filling the ventricle is greatly diminished, as in cases of mitral stenosis. Here the quantity of blood reaching the ventricle, and the force with which it enters the ventricle, are so greatly diminished, that we must look for some other cause for the dilatation of the left ventricle that is found in advanced cases of mitral stenosis.

Moreover, in acute affections of the heart, such as in rheumatic fever, we may find patients with hearts grievously affected, showing no increase in the size of the heart, while in others the dilatation may be very great. On recovery from the later condition, the heart may be left apparently undamaged. The dilatation of the heart which takes place at the onset of auricular fibrillation only occurs in some people. Other agents, such as alcohol and arsenic, may produce temporary dilatation of the heart.

It is usual to ascribe such changes to a defect in tone, and the presence of tone is said to be demonstrated in certain experiments. Following the employment of weak solutions of sodium hydrate, antiarin, and digitalin, so little relaxation of the muscle takes place after systole that the beats gradually become smaller till the heart stands still in systole. By the use of muscarin and lactic acid, on the other hand, the contractions gradually decrease in size while the heart dilates during diastole until ultimately the heart stands still in diastole (Gaskell).

It has already been mentioned that tone has been spoken of as a function of the muscle cell, but it was shown instead to be a reaction—the outcome of the response of the muscle cell to an impulse (p. 66).

Effort and dilatation.—No one who carefully watches his patients through acute and chronic illnesses can fail to recognize that there are agents which have a specific effect in diminishing the tone of the heart and permitting it to dilate. The view is held by some that when the heart is stimulated to an increased activity it dilates. It is supposed that the increased length of the fibres, which results from moderate dilatation, permits of a more vigorous contraction. Certain experiments seem to justify this view, and indeed, it finds credence all the more because it is a popular belief that strenuous bodily effort induces dilatation of the heart. I have made some observations on this matter. Thus, a healthy and vigorous young man of twenty-four was carefully placed in a particular position and an X-ray photograph taken of his heart. He then ran up and down stairs for three minutes until he was breathless and the heart's rate increased to 160 beats per minute. Immediately on stopping his effort he was placed exactly in the same position, and another

X-ray photograph taken, and the result showed a distinct diminution in the size of the heart. The same performance was gone through with a man 70 years of age, and notwithstanding his greater breathlessness, the size of the heart had not altered. The fact has been recognized also that at the end of a day's work the heart of healthy labourers is smaller than it is at the beginning of the day.

When one thinks over the matter, there is every reason for assuming that the healthy heart would become smaller. The capillaries that supply the muscles are enormous in number and capable of containing, when dilated, many times the amount of blood in the body. Muscular effort causes a dilatation of these capillaries, and consequently, a very considerable portion of the blood is in the muscles while they are being exercised. As the increased size of the heart is due to an increase in the amount of blood, it is manifest that the blood cannot both increase the size of the heart and supply sufficient to the muscles.

Dilatation and heart failure.—The reason I deal with dilatation at some length is because it is associated with certain forms of heart failure, which I am unable clearly to understand. The most typical instance of the relation to heart failure is seen in auricular fibrillation, and then it was pointed out that dropsy and enlarged liver were due to a diminished *vis a tergo* on account of the imperfect and ineffective beats of the ventricle. But we get these symptoms when the beats are not of this type, and when the rhythm is normal, although in my experience such instances are rare.

Long ago I made the observation that dropsy of cardiac origin never occurred without dilatation of the heart, and that it was very often associated with an abnormal rhythm as auricular fibrillation and flutter. The former of these observations is of some importance in differentiation. I remember attending a girl for a mild attack of rheumatic fever, and the heart was left with a systolic murmur. A few years later she began to have swelling of the feet, and I pushed digitalis till she was sick, with no effect upon the swelling, and the heart only slowed a little at occasional intervals. As the girl was getting weaker, a consultation was held with a distinguished consultant, who confirmed my diagnosis and told me to continue the treatment with digitalis. Shortly after this I went for a holiday, and on my return found my patient no better. I reviewed the whole case again, and it occurred to me that I had neglected one of my own principles—there was no increase in the size of the heart—and therefore the dropsy could not be cardiac in origin. Some months later the face began to swell, which put the cardiac origin of the dropsy out of the question. From this date she grew steadily weaker, and died, with no sign of cardiac or kidney disease.

A man was brought to me with dropsy of the legs and an enlarged liver, who had been treated for heart failure for two years. The heart was not enlarged and the rate was between 60 and 70, regular, and the pulse of good strength. The condition turned out to be one of cirrhosis of the liver.

Muscle failure.—I saw many cases of ' muscle failure ' (as Graham Steell

called it) when the beer in Lancashire was contaminated with arsenic. Amongst beer-drinkers at that time peripheral neuritis was common, and also dilatation of the heart and extreme heart failure with dropsy. Most of the cases I saw were in the Manchester Infirmary, under the care of Graham Steell, and I had only a few in Burnley, of which Observation 70 is an example; though in this instance the dropsy was not extreme, the symptoms of dilatation of the heart were marked.

There can be little doubt that the dilatation of the heart and weakness of the muscle which led to the heart failure were due to the arsenic, or the arsenic combined with alcohol. When the contamination of the beer by the arsenic was stopped, these cases completely disappeared.

A similar type of heart failure may result from a microbic infection, as in Observation 71. G. A. Sutherland has recorded the condition in a number of children in whom there were dilatation of the heart, dropsy, and a rapid heart action with the normal rhythm of rheumatic origin. These responded to digitalis in a remarkable manner, the pulse-rate becoming slow with disappearance of the dilatation and dropsy.

Occasionally we meet with patients who give a history of rheumatic fever, and who have been left with disease of the mitral valve, who break down with dilatation of the heart and extensive dropsy. They may become gravely ill and yet after months make a good recovery, and afterwards lead an active life for many years, free from any sign of heart failure.

Most patients with muscle failure and dilatation do not have dropsy, but suffer from difficulty in breathing—orthopnœa, Cheyne-Stokes breathing, cardiac asthma, sometimes with more or less pain in the chest. It is in this type of patient that the pulsus alternans occurs and is of ominous significance (Observations 69, 72, and 73).

The features are seen in the observations recorded in the next chapter.

It is sometimes remarkable how far disease may proceed before any sign of its presence is revealed by an impairment of the functional efficiency of the heart. One of the most remarkable instances is recorded, 'Observation 74', where a farm labourer continued at his work unconscious of any limitation till within three weeks of his death. The damage due to the gumma did not impair the efficiency of the heart till it had advanced a long way.

Prognosis.—It will be gathered that the symptoms of heart failure due to myocardial affections are common to all forms of structural changes. To obtain a prognosis it is therefore necessary to form some idea of the nature of the myocardial change. If due to a poison, whether rheumatic or microbic in origin or from alcohol or arsenic, its prognosis depends then on the cessation of the poisoning or on combating its effect. When the failure is due to functional disturbance, and the response to treatment satisfactory, the prognosis is favourable. But where the muscle is damaged, then the prognosis will depend on the extent of the damage, and this will best be estimated by the manner in which the circulation is maintained when the body is at rest, and when effort is made, after treatment has been tried.

The appearance of Cheyne-Stokes respiration is not usually of grave significance by itself, for many people getting on in years exhibit it and still lead useful and even fairly active lives. But when it reaches the degree which produces these severe attacks of breathlessness in the night—cardiac asthma—then the condition becomes grave, many dying within a couple of years of its first appearance. It is not so grave when the failure is associated with auricular fibrillation and the heart responds to digitalis and rest. But when the normal rhythm is present and the patient on in years, the outlook is grave, especially if associated with the pulsus alternans.

Treatment.—Whenever the heart failure has reached the stage to cause breathlessness on slight exertion, orthopnœa, dropsy, or enlarged liver, rest is imperative. The heart failure may have been brought about by the progress of the disease or the intensity of the poison, but in either case it has been hurried on by the patient undertaking more effort than the heart was capable of performing efficiently, so in taking rest one factor is removed.

The rest does not require putting the patient to bed ; he may be better sitting up. The kind of rest is that in which the patient is free from distress. Many patients are far more comfortable in an easy-chair than in bed. Some can sleep thus, or sitting up with the head leaning forward on a table or other support.

When the condition is not so far advanced, and the patient can walk a little without discomfort, he should be allowed this liberty, any form of distress on walking being a sign that too much effort is being made.

Without knowing why or wherefore, drugs of the digitalis group have occasionally a good effect (see Observation 69), so these should be tried in every case. In the same way the different methods of increasing the flow of urine should be tried when there is dropsy. All factors that tend to disturb the patient should be removed, with careful attention to the diet.

In these cases as in the elderly, the irremediable condition being recognized, there is no use in attempting ' cures '. It is better to recognize the fact and then proceed to make the life of the patient as comfortable as possible, soothing any irritation, helping sleep by the milder hypnotics, placing the patient in as agreeable surroundings as the circumstances permit, avoiding unnecessary expenses when the financial condition of the patient, or those on whom he depends, requires economy.

CHAPTER XLVIII

Cases illustrative of Affections of the Myocardium

Observations 69-73.

OBSERVATION 69.—Male. Aged 66. Retired schoolmaster. Seen January 12, 1920. Complains of breathlessness on exertion. Breathlessness had been gradually coming on during the last few weeks.

He had enjoyed good health, but before he retired four years ago he was becoming weak and irritable, but has been much better until the last few weeks. The patient is stout and healthy-looking, the pulse regular, 120 beats per minute when lying down. Systolic blood-pressure 180, diastolic 140 mm. Hg. The heart's dullness is increased in size, the apex is in the 6th interspace, and the dullness is 2 inches outside the left nipple and 1½ inches to the right of the middle line. The sounds of the heart were free from murmur, but there was a cantering rhythm.

28.1.20. Under tincture of digitalis he speedily improved, the breathlessness practically disappeared, and he is now walking up hills with comfort. The pulse-rate has fallen to 86 (Fig. 249). Blood-pressure is 200 systolic and 140 diastolic.

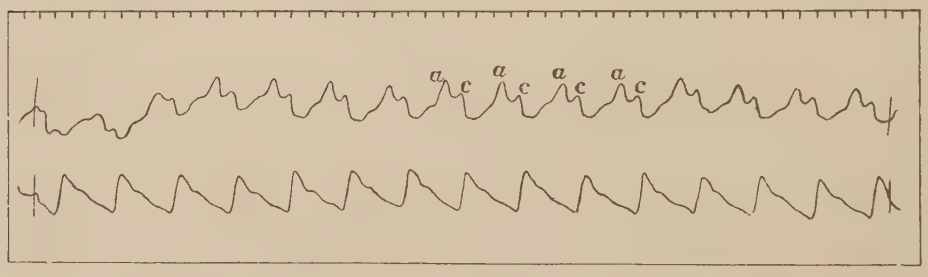


Fig. 249. Shows the normal acting heart in 1920. (Observation 69.)

He still continued improving, though the blood-pressure varied from 190 to 205 systolic, and from 120 to 160 diastolic, the rate of the pulse being usually about 80 beats per minute, and sometimes 100. The heart's size was not perceptibly affected. The urine showed no albumin, but there was a small quantity of glucose which varied from 0·55 per cent to 3·4 per cent. In April 1922 he reported keeping well, golfing and gardening. In October 1922 he stated he had not felt so well for a long time as during the last eighteen months, and enjoyed playing a round of golf. The heart is still the same size; there is no canter rhythm, the rate while lying 108 per minute B.P., 190 syst., and 140 diast. A trace of protein in the urine and also sugar. The radial tracing showed a slight pulsus alternans. He was seen occasionally during 1923, but on December 20 he had become very breathless again, and was only able to lie in bed with comfort so long as his head was raised on a fairly high pillow. He was totally unable to play golf because of the breathlessness; there was a good deal of cyanosis of the cheeks and ears; the pulse was 120 per

minute, with an occasional extra-systole and very marked pulsus alternans (Fig. 250), B.P. 190 with large beats, and 170 for the small, diastolic pressure 120. He was put upon digitalis, 15 drops three times a day, and in four days' time he felt remarkably better, although very little difference could be detected in the size of the heart or rate of the pulse, except that the pulsus alternans was not so marked (Fig. 251), the blood-pressure being 190.

In July 1924, though somewhat short of breath, he felt remarkably well.

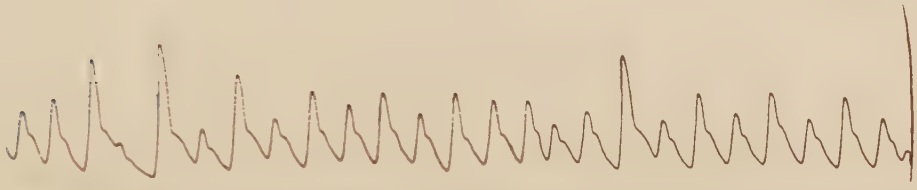


FIG. 250. Taken during a temporary breakdown, showing the presence of extra-systoles and the pulsus alternans. (Observation 69, December 20, 1923.)

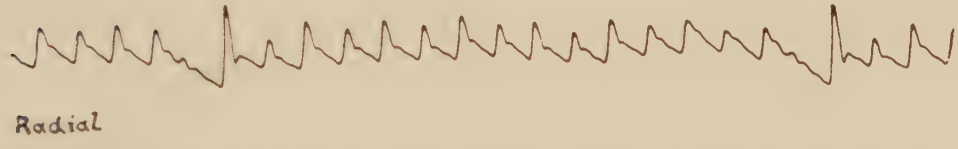


FIG. 251. After digitalis there was great improvement in the condition, and the pulsus alternans was much less marked. (Observation 69, December 24, 1923.)

OBSERVATION 70.—Aged 37. I had this patient under observation for two years, generally for some discomfort after a drinking bout. I saw him on May 15, 1902, complaining of pain across the chest and difficulty in breathing. He had been drinking rather heavily of late and consuming a good deal of beer. He had been for a walk into the country on the morning of the day I saw him, and had taken rather a hilly road and had been seized with the pain and distress in breathing. When I saw him he was in bed with his shoulders raised, tossing from side to side because of the pain in his chest, which was not severe, but sufficient to prevent his lying down in comfort. His eyes were slightly jaundiced, and the radial pulse was large, full, and compressible, 130 beats per minute. The apex beat was diffuse and out beyond the left nipple line, and the dullness extended 2 inches beyond the nipple line on the left and ½ inch to the right of the middle line. There was a loud, blowing systolic murmur, heard loudest over the third left costal cartilage. The veins of the neck were full and pulsated largely, the waves ascending as high as the angle of the jaw. The neck was long and the veins stood out, and there were two waves (*a* and *v*) to each beat of the radial pulse, the neck thus presenting the appearance of a continuous movement (Fig. 252). The liver was slightly enlarged, and the subcutaneous tissues covering it (skin and muscle) were very tender on pressure.

With rest and sedatives and abstinence from alcohol the heart symptoms gradually subsided, the rate became slow, the pulsation in the veins of the neck disappeared, and ten days after the

patient was much improved (Fig. 253). The dullness decreased, the heart's apex was inside the nipple line, and the systolic murmur was almost gone.

After this the patient's progress was uninterrupted, and he went on well for several months till he began drinking again, and nine months after he had another attack of heart failure, which followed a very similar course, and was again followed by recovery on abstaining from alcohol. At this time it was discovered that the beer was contaminated with arsenic, and this patient stopped the beer-drinking. A year later he developed delirium tremens. He had not been drinking beer, but whisky and brandy. This time there was no dilatation of the heart nor pulsation in the veins of the neck, though the pulse was rapid. The patient died from exhaustion.

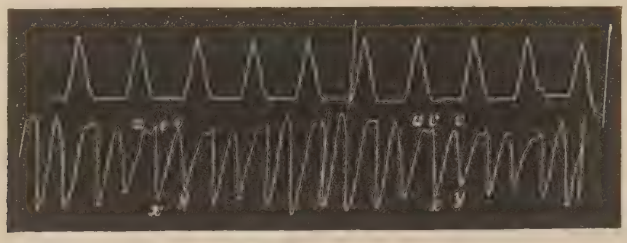


Fig. 252. Simultaneous tracings of radial and jugular pulses, to show the large jugular pulse while the heart was poisoned. (Observation 70.)

Fig. 253. Taken 10 days after Fig. 252, to show the difference in the character of the radial and jugular pulses in recovery. (Observation 70.)

OBSERVATION 71.—Aged 53. The patient was sent to me by Sir A. Wright on April 14, 1911, because of the condition of his heart. On walking across my consulting-room the patient was very breathless, and had to sit a few minutes before he had breath to speak. He complained of pain across the chest and breathlessness on the slightest exertion. He was very strong and well up till two years ago, when he had a double pneumonia, and since that time he has been extremely weak and breathless, and has been getting gradually worse. Sir A. Wright reported that he had still a streptococcal infection of the lungs, which he proposed to treat by vaccines. The patient's face had a jaundiced tinge and was slightly livid. The pulse was large and soft, 70 per minute. The heart was dull 1 inch beyond the nipple line on the left. The sounds were clear, and the jugular veins were full and pulsated largely.

The picture presented by the patient so strikingly resembled that of Observation 70 that I concluded that the patient's heart was being poisoned, as in that case, by the toxins of some microbic infection, so I sent him back to Sir A. Wright with this view, and suggested that his treatment by vaccines would be far more likely to be successful than anything I could suggest. The vaccine treatment was employed. By October the patient had improved so greatly that he was able to go shooting. Two years later I heard that he was leading an active life, and free from any heart trouble.

OBSERVATION 72.—Male. Aged 72. I had known this patient for over twenty-five years as a sober and industrious man. He worked as an engineer to within a year of his death. He had a slight attack of hemiplegia in December 1906, from which he made a good recovery. He consulted me in June 1907 because he passed blood in his urine. Except for being rather short of breath he felt fairly well. He looked a hale old man. His radial arteries were large and tortuous, pulse full, seemingly regular. His heart's dullness extended to the nipple line. The sounds of the heart were clear except for a musical murmur, systolic in time, heard over the whole heart region, but loudest in the aortic area. It varied distinctly in loudness, a loud murmur alternating with one less loud. When I took a radial tracing it showed a well-marked pulsus alternans. He visited me several times. The urine became quite clear, but the heart's condition continued, and extra-systoles were sometimes very frequent, and gave to the pulse the appearance of extreme irregularity.

Early in August he complained greatly of attacks of bad breathing in the night. He would go to sleep quite quietly and then awake suddenly with a sense of suffocation, and sit up in bed breathing heavily. After half an hour he would feel easier, but could not lie down, and had to be propped up in bed. These attacks occurred on several occasions until they disappeared in September, when his legs began to swell, and he expectorated quantities of blood-stained mucus,

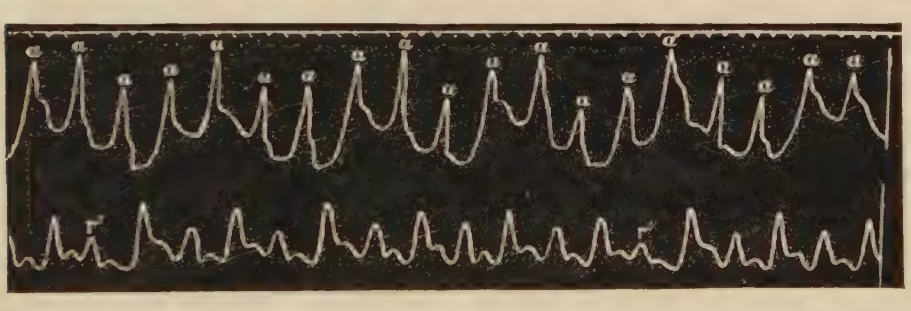


FIG. 254. Shows the pulsus alternans in the radial with ventricular extra-systoles (*r'*). In the jugular there is only one large wave (*a*), due to the auricle. (Observation 72.)

and small clots of blood. His heart's dullness extended 2 inches beyond the nipple line; the veins of the arm became full. Venesection was tried, but with little good result, and he died in October.

Report of the P.M. examination of the heart.—The mitral and tricuspid valves show no pathological changes. The auricles are not markedly dilated; the coronary sinus is filled with post-mortem clot. The ventricles are not dilated, except at the apical part of the left. Both coronary arteries show a thickening of their coats with dilatation, the left more than the right, and the anterior inter-ventricular branch the most affected. At the apex of the left ventricle is an area showing intense fibrosis, with deposits of premature clots; the microscopic sections show the Purkinje and inner muscle-layer healthy. The a.-v. node and bundle show a fibrosis, not intense, and also a stretching of the bundle, as if the pars membranacea had been stretched. There is no sign of cellular change in the bundle or node except the predominance of the fibrous tissue over the muscle-tissue, the muscle fibres, instead of reticulating, being stretched and parallel.

OBSERVATION 73.—Male. Born 1843. I had attended this patient for an attack of erysipelas in 1880, following on a wound on the head. He made a good recovery and led a healthy, active life until 1890, when he began to drink heavily at intervals. He would abstain for many months, and then would drink heavily for a few weeks. After 1900 these outbreaks occurred three or four times a year. During his drinking bouts his heart became rapid in its action and dilated, and it was generally because of the extreme prostration that he stopped drinking for a

time. When he ceased drinking, in a few weeks' time his heart would be quite normal, and there was no limitation in its power. After rather a long bout in 1907 he pulled himself together, and ceased to take alcohol except in limited quantities.

In May 1909, then aged 66, he consulted me, as he was feeling the heart thumping at times, though he was able to go about his affairs. He was taking no exercise and was getting stout. The pulse was hard, the blood-pressure 210, and there were frequent extra-systoles followed occasionally by a very slight pulsus alternans. The heart's dullness extended to the left as far as the nipple line, and the sounds were clear. There was no albumin in the urine.

On March 29, 1910, he was feeling fairly well, and could walk about in comfort. The pulsus alternans was well marked, and the blood-pressure for the large beats was 180. On October 18, 1910, he reported himself as doing well, though rather short of breath.

About the middle of February 1911 he felt slight distress at times across the chest, but the breathing was easier, and there was only slight pulsus alternans. In April of this year he reported that he was feeling weakly and had been so for a fortnight, and that he had a dragging sensation over the heart. He was sleeping badly and felt squeamish and sickly about 3 A.M. He had wakened up early in the morning, feeling breathless. The heart's dullness was 1 inch outside the left nipple line. Six weeks later he was seized with an attack of intense dyspnœa during the night, which lasted four or five hours, and during which he went very blue and was scarcely conscious. He recovered from this and went on fairly well. I saw him on June 17; he had been troubled at night with the breathing, though there were no very bad attacks. There was an account from his doctor of an attack of distress (not with bad breathing), when his pulse-rate was nearly 200 per minute. He had also had Cheyne-Stokes respiration. He was lying comfortably in bed when I saw him. The pulse was rather frequent, 110 per minute, and I could detect the pulsus alternans very slightly and for short periods. The heart's dullness now extended 2 inches beyond the nipple line, and the apex was diffuse and rather forcible.

After this the patient gradually went downhill, with attacks of dyspnœa during the night, and he died on August 19, 1911.

CHAPTER XLIX

Syphilitic Affections of the Heart

As every tissue in the body is liable to be affected by syphilis, the organs of the circulation are attacked in a variety of ways. The arteries have long been known to be peculiarly liable, as in the production of aneurysms and of degenerative changes in the smaller arteries, affecting particularly those of the brain and the heart muscle. In addition to the production of aneurysms of the aorta, syphilis is frequent in the first part of the aorta (syphilitic aortitis), where not only the aorta is damaged, but the valves, and the opening of the coronary arteries are involved, giving rise to a variety of symptoms. The muscle fibres are not immune, though perhaps the damage may be secondary to the arterial disease. Gummata, too, may occur in the ventricle, but rarely give any distinctive sign unless they invade the genetic system and produce heart-block (Observation 74).

The presence of angina pectoris, aortic regurgitation, or heart-block should always lead to a close inquiry into the possibility of syphilis being the primary agent in producing the diseased state.

It may be as well to point out that because a patient may have had syphilis it is not wise to assume that thereafter every sign of disease is syphilitic in origin and calls for active treatment. I have repeatedly seen patients with an extra-systole, or a systolic murmur who admitted having syphilis, alarmed by the diagnosis of a syphilitic affection of the heart. They have willingly submitted to salvarsan injections, with no alteration in their state. Indeed, though I have seen many elderly patients who gave a positive Wassermann reaction rigorously treated by anti-syphilitic remedies, in no instance did any improvement follow. Of course it can always be said that the progress of disease might be checked. While that is problematical, there is no doubt of the actual harm often done by alarming the patients unnecessarily.

The injudicious manner in which the recent methods of anti-syphilitic treatment are so widely used threatens to do much harm. While the employment of 606 and its modifications holds out the prospect of successfully combating the disease, it is far too soon to say of the treatment that it is curative. It should be remembered that many people have had a syphilitic infection and show no signs in after life, with little or no treatment. Many people show

signs only after a long period of years, and because the immediate symptoms disappear under treatment, it does not follow they will not suffer in later life, even if they do not give a positive Wassermann reaction. Experience already shows the recurrence of syphilitic symptoms after most careful treatment, even when the Wassermann was reported negative. It must also be borne in mind that the Wassermann reaction is not to be relied upon, for not infrequently one expert may say a patient's blood is positive while another reports it negative. Whether that is due to the method or the expert, I cannot tell.

My object in writing this is to warn against the assumption that any cardiac sign in a patient with a history of syphilis is due to syphilis, and to draw attention to the need of a long and patient investigation into the treatment of patients when the disease can with fair certainty be assumed to be syphilitic. I do not deal with the matter at length, because my experience does not enable me to throw much light upon the subject.

OBSERVATION 74.—Male. Aged 53. Examined 5.8.20.

July 1920.—This man enjoyed good health and was doing the work of a ploughman regularly, and without inconvenience, until the second week of July 1920. About this time he began to feel a dull aching pain in his chest just after retiring to bed; it usually eased off after an hour or so, and was not present during the day. There was no palpitation, faintness, or giddiness. He continued at work during the last three weeks of July, and for the first time, on August 1, there was breathlessness on exertion.

August 5.—Examined five days later his heart was found to be enlarged and there were double aortic murmurs, loudest at the base, but heard down the sternum also. His pulse was 60, regular, and his blood-pressure 140 mm. Hg. During the following week his condition became gradually worse, his colour became ashen, and his ears cyanosed, and he was compelled to sit up in bed.

August 12.—On August 12 there was tenderness in the epigastrium; his liver and spleen were enlarged; his pulse was 44 per minute; there was no œdema. He complained greatly of thirst. A polygraph tracing showed a condition of complete heart-block. He had Cheyne-Stokes respiration, and there were repeated attacks of cardiac asthma. There were no syncopal attacks; $\frac{1}{60}$ grain of atropine sulphate produced no recognizable effect; 1 drachm of tincture of digitalis was given every twenty-four hours, and this produced a temporary amelioration of symptoms, but was stopped on the fifth day, when the ventricular beats were coupled.

August 19.—The symptoms were much aggravated by this time; Cheyne-Stokes respiration was still present, and during an attack of dyspnœa the pulse rose for about 20 beats to a rate of 100 per minute, reverting after the dyspnœa to 42 per minute.

August 21.—During the following two days he became more breathless, was sleepless, and complained of severe pain below the ribs in front on each side, and especially on the right side in the liver region. His face became puffy and his hands and feet swollen and œdematous. The pulse ran between 40 and 42 beats per minute, with an occasional interruption of irregularity intervening in the heart-block.

August 22.—He died on August 22, three weeks after the breathlessness began, and six weeks after the first appearance of discomfort in the form of evening pain.

Post-mortem examination, by Dr. Ivy Mackenzie (Figs. 255, 256, and 257).—A well-developed, muscular man. There was general œdema, a small quantity of fluid in the peritoneum, pericardium, and pleural sacs. The solid viscera showed signs of passive congestion. The heart was enlarged and weighed 18 oz. Before opening the heart an aneurysmal dilatation of the aorta was noticed. It was situated in the right posterior wall of the lower part of the aorta

and was large enough to occupy half a tangerine. Dilatation of the base of the heart revealed a hard thickening in the front and to the right side of the region of exit of the pulmonary artery and aorta. The four chambers of the heart were abnormally dilated and contained a considerable amount of ante-mortem clot.

Two large areas of gummatous infiltration were noticed in the musculature of the *right ventricle* (see Fig. 255, right ventricle); one moderately large and about the size of a walnut lay in the anterior wall of the infundibulum and extended from the moderator band below to the

FIG. 255. Interior of right ventricle, showing the projection of the gummatous mass on the septal wall.

membranous septum above and behind; the other, more diffuse, infiltrated the wall of the interventricular septum and could be seen beneath the endocardium extending in the infundibulum from the insertion of the pulmonary valves right through the moderator band to its insertion in the anterior wall of the right ventricle.

In the *left ventricle* the diffuse gummatous infiltration could be seen and felt in the interventricular septum throughout the region in which the left limb of the auriculo-ventricular bundle begins to spread. The remainder of the muscle substance was soft and flabby (Fig. 256). The aortic valves were incompetent, as were also the tricuspid and mitral, through disproportionate dilatation.

Aorta.—The thoracic aorta was diseased throughout its whole extent. There was extensive cicatrization and distortion typical of syphilitic disease. The right posterior wall showed the aneurysm which was observed before the heart was opened (Fig. 257).

On cutting the *musculature* the gummatous infiltration was seen to be confined more or less to the areas corresponding to the surface appearances already described. It was possible to trace the gummatous degeneration from the aortic wall into the musculature of the inter-

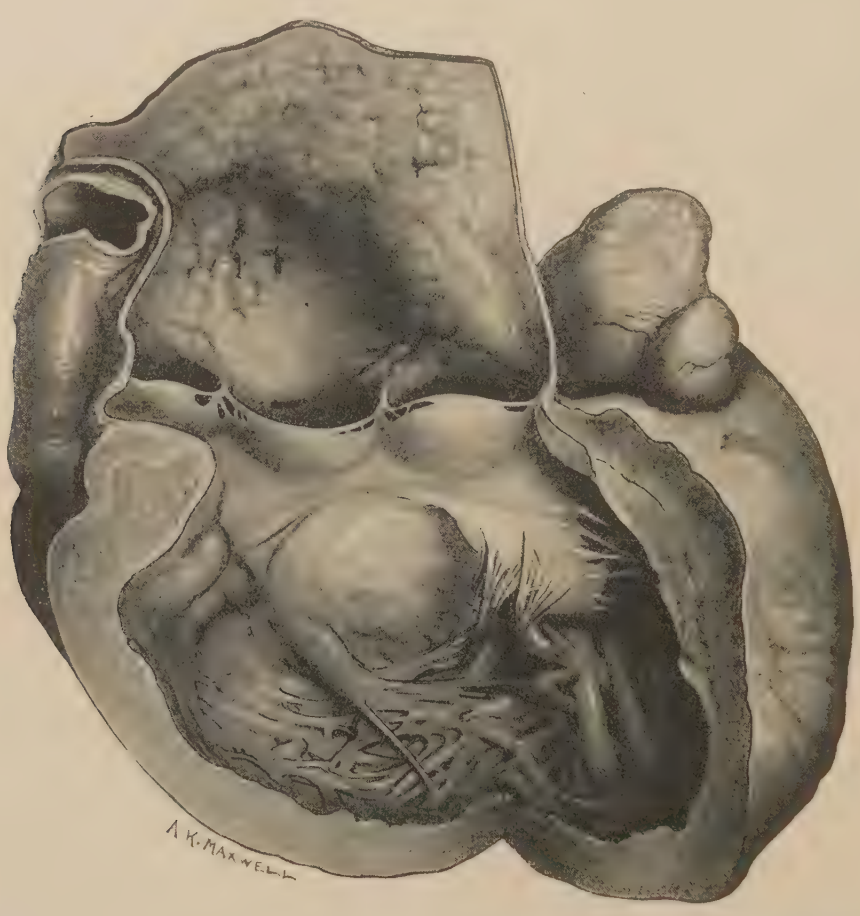


Fig. 256. Interior of left ventricle and of aorta, showing the projection of the gummatous mass into the interior of the left ventricle.

ventricular septum, and already one could see from the site of the infiltration that both limbs of the auriculo-ventricular bundle must have been involved.

Microscopic section of the diseased muscle indicated a recent and acute phase of syphilitic degeneration and infiltration. There were large areas of necrosis, and round these areas of inflammatory reaction. The most extensive degeneration was found in the region of the inter-ventricular septum where the two limbs of the bundle descend to their distribution. The bundle itself, however, was infiltrated in its main course, although at no part in its main course could it be said that there was total interruption of the muscle fibres by disease.

FIG. 257. Section of the interventricular septum, showing the gummatous mass occupying the upper part of the muscular septum, below the membranous portion, in the position of the left limb of the a.-v. bundle.

CHAPTER L

Angina Pectoris

Introduction.
Vital processes.
Stimulation by mechanical means.
Stimulation by vital processes.
Impulses from muscles.
Pain from muscle.
Stimulation.
Hyperalgesia.
Experimental hyperalgesia.
Increased excitability of the nervous system.
Two forms of angina pectoris.
The localization of pain.
The value of pain in diagnosis.
The value of pain in prognosis.
Angina pectoris and the principle of the reflex arc.

Introduction.—Angina pectoris has been a subject to which I have devoted a great deal of study. The results of this study have been published recently in a book,[1] and in it there is given in detail a description of the views I have come to, together with a large number of illustrative cases.

There is no matter which so reveals the poverty, or even the bankruptcy, of modern medicine in conceptions than the subject of pain to-day. It is of all symptoms the most clamant, the most universal, and probably the most diagnostic, and one calling, above all others, on the resources of the doctor. It has long been a subject of investigation in which great numbers of the most skilled have participated, and yet to-day there is little beyond vague speculation, neither the structures nor the vital processes concerned in its production being recognized. The depressing part about the matter is that it is not known what steps should be taken to acquire this knowledge.

During the many years that I have been engaged in the investigation of pain it has been possible to make only slight progress, for most of my attempts were rendered abortive, because there is nowhere to be found the elementary knowledge necessary for the investigation.

My observations, therefore, were limited to noting the circumstances governing its onset and, as far as possible, the detection of the source of the disturbance which induced it, and any other phenomena associated with its presence.

[1] *Angina Pectoris.* Oxford Medical Publications, 1923.

In the course of this investigation I recognized, in 1891, the presence of cutaneous hyperalgesia, and found that this sign was one of the greatest value in getting that knowledge of sensory disturbances necessary to the practice of medicine. Following this I found another phenomenon, namely, a contraction of limited portions of the muscle of the abdominal wall in visceral disease (the viscero-motor reflex). In the course of investigation into these phenomena I gradually saw the possibilities of obtaining a better insight into the principles that underlay the production of symptoms. It was from such studies that there gradually emerged the view of fluctuation, of the meaning of tone, and of the character of the cell impulse, and of other matters described in Chapter XII.

In the following description some portions of this research are utilized, but the subject is still far from being exhausted, and there is waiting an excellent field for research.

Vital processes.—It has been insisted all through this book that all reactions, whether sensations, contractions of muscle, or secretions of glands, are due to the participation of vital processes and living structures, and that vital processes are based on the excitability of the cell and the impulse.

While we can recognize some of the structures, there is but a hazy notion of what is meant by a vital process. Like all forces in nature, a living process is only recognized by the effects it produces. By the observation of these effects we can recognize that there are vital processes which result in the discharge of impulses, in their conduction, and in their producing an effect such as the discharge of function by the cells they reach. There are, again, vital processes concerned in rendering the cell susceptible to the impulse—what is called excitability.

It is incumbent, therefore, to look at the vital processes which originate the impulses concerned in producing pain, as no reaction can take place except by an impulse stimulating a cell.

Stimulation by mechanical means.—Mechanical stimulation to the skin and other structures supplied by the cerebro-spinal sensory nerves can result in the production of pain. All that can be said at present is that a mechanical stimulation of the cells of the skin and certain other tissues produces impulses which, when conveyed into the central nervous system, result in the stimulation of a peculiar group of cells which gives rise to the sensation of pain.

Stimulation by vital processes.—There are structures which are not supplied with cerebro-spinal sensory nerves, but by nerves of the autonomic system. These are totally insensitive to mechanical stimulation, but are sensitive to certain vital processes which result in the production of pain. It can be inferred that while the afferent nerves of the cerebro-spinal system are adapted for the reception of impulses produced by a mechanical stimulation, the afferent nerves of the autonomic system are specially adapted for the reception of impulses produced in a different manner, just as the retina is specially adapted for the reception of impulses produced in a particular way.

As we are dealing in this book with visceral pain, *i.e.* pain arising from

impulses which have been taken up by the autonomic nerves and are produced by some vital process, it is necessary to recapitulate briefly some results of the investigation into the nature of this vital process.

Impulses from muscles.—In the description of the generation of impulses it has been shown that one great source of the production of impulses is the muscular system. This has been shown by the contraction of voluntary muscles producing impulses which increase the heart-rate (p. 95), and by the impulses produced in auricular flutter, stimulating the a.-v. node (p. 45). It is shown in the contraction of the parturient uterus, producing impulses which give rise to a great number of reactions. Among these reactions is that of pain. There is thus abundant evidence to show that the muscle in contraction at the same time discharges impulses which can give rise to pain. It is also shown that the fibrils of the afferent nerves of both cerebro-spinal and autonomic systems are specially adapted for the reception of these impulses.

Pain from muscle.—If a wide outlook be taken over the whole muscular system, pain will be found to be so commonly associated with this contraction that there is good ground for assuming that there is a definite relation between pain and the contraction of muscle under particular circumstances.

A man had a blocked left femoral artery from which he recovered, but the leg was somewhat wasted. On resting and when walking quietly he had no distress, but if he walked rapidly, after a short distance the aching in the left leg became so severe that he was compelled to stop. A man found one morning that he was unable to walk more than fifty yards because of pain in his right leg and foot which compelled him to stop. On examination the right foot was blanched, and no pulsation could be found in the arteries of his foot; evidently one of the arteries of his leg had become blocked. A woman suffered from attacks of auricular flutter, and on several occasions the cessation of attack was followed by embolisms blocking up certain arteries. On one occasion the doctor found the left arm cold, and there was a total absence of the radial pulse. The brachial artery had been blocked by a clot from the heart. When the patient firmly shut her hand and opened it several times, great pain was felt in the arm, so that she was compelled to stop. MacWilliam has shown that if the arm of a healthy man be deprived of blood, the forced contraction of the muscles of the arm will produce pain. A man, 54 years of age, had occasion to walk four miles hurriedly to catch a train, and he discovered after about three miles' walking that he had the greatest difficulty in moving his legs. Persisting in the attempt resulted in such pain that he was compelled to stop. As years went on this condition occurred earlier, so that after walking a quarter of a mile a sense of weakness and aching in the legs and feet compelled him to stop. On several occasions on walking and stopping for a distance of several miles, some hours after, when resting, attacks of pain of great severity would be felt in his calves, but lasting only a few seconds. At the age of 70 he began to be pulled up when walking shorter distances by pain in his chest, and he suffered no more from pain in the legs, because he was compelled to stop walking by the pain in his chest before the

PLATE I.

Heart viewed from the left side. (Slightly enlarged.)

The left coronary artery has been exposed, and shows nodular thickening and calcification of the wall in patches.

legs were affected. A man of 56 complained of being pulled up when walking a quarter of a mile because of pain in his feet. Some months later he began to be pulled up on walking by pain in his chest, so that at one time his powers of walking were limited by pain in his chest, and at another time by pain in his feet. Latterly, for six months before he died, he suffered no more pain in his feet, but only in his chest, which became so readily provoked that it appeared after walking twenty or thirty yards.

Pain, associated with muscle, can be brought about in other ways than by exertion. Thus, a man who had hitherto suffered from pain in his chest (angina pectoris), on walking found that the pain could be produced by a curious vaso-motor reaction. He found that on going to bed, when he got between the cold sheets he shivered from cold, and pain in his chest resulted. He had always enjoyed a cold bath, and he experienced a pleasurable glow when he dressed, but he had to give this up, because about half an hour afterwards a reaction set in, when he felt cold and shivery and a severe attack of pain came on. He tried a hot bath, which he enjoyed, but after dressing his hands went cold, he shivered, and a violent attack of pain came on. These attacks were all relieved by amyl nitrite. One of my colleagues suffered from a sudden febrile attack such as occurs in cholecystitis. Before the temperature rose he had a sense of profound prostration, and at the same time the fingers of both hands became blanched and cold. On walking and on going upstairs he had to stop because of pain in his feet, which disappeared as soon as he stood still. On taking off his socks preparatory to getting into bed he found both feet cold and blanched, like his fingers.

It is not clear how vaso-motor reactions such as these produce pain. If the view that impulses from muscle are the cause be established, it would seem that the vaso-motor contraction extended to the vessels of the heart. The heart's contraction continuing with the diminished supply of blood might be the cause. In support of this view is the fact that the pain provoked by voluntary muscles just described only occurred when the muscles contracted.

In the investigation into visceral pain, every instance of severe abdominal pain whose origin could be recognized has been found associated with a hollow muscular organ, as in renal and gall-stone colic, in colic of the bowel, in contraction of the uterus.

Plates I.-IV. are drawings of the hearts of a series of patients, and they all show damage which diminishes the supply of blood to the heart muscle, as in the blocking of coronary arteries, or damage to the muscle itself in consequence of the diminished blood supply. The chief and often the only sign to which the damaged heart gave rise during life was anginal pain in response to effort.

Stimulation.—Pain is the result of *stimulation* of certain structures. In the description given of stimulation it was shown that the vital process of stimulation was due to an increased excitability of the cells. This is brought about in different ways — by an increase in the number of impulses or by other conditions (as heat, toxic agents, loss of control). Different forms were

illustrated by reference to a labour pain, which is due to an increase in the number of impulses. Another form of stimulation was illustrated by the a.-v. node in auricular flutter, shown in Figs. 7-9, where the stimulation was shown to be due to an increased excitability of the cells of the a.-v. node, due to the loss of the control of the s.-a. node.

These forms of stimulation are to be found in the production of pain, and other reactions or symptoms.

Hyperalgesia.—In some people who have a decayed tooth the skin of the cheek may become so sensitive that the stroking of the hairs of the cheek will produce pain. Hyperalgesia of the skin and of the deeper structures of the abdominal wall is a common sign in disturbances and diseases of the abdominal organs, such as the enlarged liver which occurs in heart failure, appendicitis, gastric ulcer, and after attacks of renal, bowel, or gall-stone colic.

There is no change in the skin and deeper tissues in these circumstances; the hyperalgesia is the result of some portion of the central nervous system which has been rendered hyperexcitable, so that impulses which usually produce a sense of touch or pressure now produce that combination which results in pain. Hypersensitive areas in the central nervous system can result in the production of a great variety of phenomena. An individual who is sickening of some febrile attack or who is having an attack of migraine may be extremely susceptible to influences which may produce vomiting. A whiff of tobacco smoke, an unpleasant sight or a loud noise may induce an attack of vomiting. Other people under certain conditions may have the functions of other organs disturbed, as in the occurrence of asthma, of diarrhœa, of frequent micturition.

Experimental hyperalgesia.—Barenne[1] has lately shown that on the application of a solution of strychnine to a minute region of the surface of the cortex of the brain of a monkey, the structures in certain regions of the external body wall become hyperalgesic. These areas seem to be of the same kind that are found in the human body as a result of disturbance or disease of the viscera.

Increased excitability of the nervous system.—There are conditions of ill health in which the central nervous system, in place of having limited areas rendered hypersensitive, shows a general hypersensitiveness, so that impulses which normally produce no perceptible reaction produce reactions that are readily perceived, as in that condition which is unfortunately called 'neurasthenia'. This is a term applied to a condition which nobody understands, and it is not a disease of the nervous system any more than the hyperalgesia of the skin is a disease. In such individuals with this hypersensitive state of the nervous system, the normal impulses which are generated by the different organs and normally pass unperceived may result in producing demonstrable reactions. As these reactions can be traced to a certain organ, then the complaint is either attributed to that organ, or the disease is spoken of as neurasthenia of that organ. When there are stomach symptoms present

[1] *Proc. Roy. Soc.*, Series B, vol. 96, pp. 272 *et seq.*

the condition is spoken of as gastric neurasthenia. If cardiac symptoms are present it is described as cardiac neurasthenia.

Among the symptoms or reactions which are produced by a healthy heart sending impulses into this hypersensitive central nervous system is that of pain. This pain may be of equal severity with, and resemble in every particular, the pain which arises from damaged hearts, indistinguishable from what is called angina pectoris.

Two forms of angina pectoris.—We have thus two forms of cardiac pain which are produced in different ways, and which have a significance of a widely different kind. The one form is associated with actual disease of the heart. This, I suggest, should be called ' primary angina pectoris '. In the other form there is no disease of the heart, the symptoms being referable to a hypersensitive central nervous system. This form I call ' secondary angina pectoris '.

The localization of pain.—A reaction like pain can be considered from various points of view. Here it will be considered chiefly from two aspects which are of value for the practice of medicine—diagnosis and prognosis. For these purposes the localization of pain has first to be considered.

With pain there is always an accompanying phenomenon, which can scarcely be called a sensation, but which is of very great importance, and that is localization. One cannot conceive of a sensation like pain without localization. Localization of pain is always associated with the peripheral distribution of cerebro-spinal nerves. On account of pain and its localization we can recognize the peripheral cerebro-spinal nerves which have been stimulated. In the central nervous system these are related to afferent nerves from the organ which produces the impulses which result in pain. This affords the means of recognizing the nerves which take part in producing pain, and in this way we obtain a knowledge of the afferent nerves which are supplied to the organs.

Other phenomena afford us the opportunity of obtaining this kind of knowledge; the chief of these is the areas of hyperalgesia which occur in the skin and other tissues of the external body wall and of the contraction of the muscles, particularly of those in the abdominal wall (the viscero-motor reflex). The peripheral nerves which are associated with these phenomena can be traced into the central nervous system, where they are in direct communication with the afferent nerves of the organ which gives rise to the increased impulses.

The value of pain in diagnosis.—There are a great number of other phenomena which may accompany pain, all of which are provoked by the contraction of a hollow muscle like the uterus. It is to be observed that all these phenomena, such as flushing, sweating, increased heart-rate, nausea, vomiting, etc., give no indication of where the impulses that provoked them arose. Patients exhibit these symptoms who suffer on account of disturbances of a great many organs, such as all the hollow viscera, the eye-ball, and the brain. It is usually a matter of hopeless conjecture to recognize the source, if pain

or cutaneous hyperalgesia or contracted muscle is absent. If one or other of these is present it affords evidence which directs us unerringly to the seat of disturbance.

As our knowledge increases we obtain a clearer insight into the different structures which are concerned in the production of pain. The position of pain in angina pectoris, for instance, gives information concerning the vagus and sympathetic afferent nerves. We know that the sympathetic nerve to the heart passes out through the central nervous system at the level of the lower cervical and upper dorsal regions. We know also that pain is felt in the region of the peripheral distribution of the cerebro-spinal nerves, which come from this region. We also know that the impulses which produce the pain in this region arise in the heart. We can therefore safely infer that such pain is an evidence of the presence of afferent nerves from the heart.

From the heart also pain is felt in other regions, along the lower jaw and behind the ear. The nerve supply of this region is from the fifth cranial nerve, and second and third cervical. We know that the vagus nerve arises in the central nervous system at the same level as those nerves; we can therefore safely infer that the pain in this region is due to impulses which are conveyed to it by afferent nerves of the vagus.

From these facts we are able to construct a diagram of the nervous system concerned in the production of the pain in angina pectoris. We are also able to correlate the nerves which participate in producing pain in this region (see Fig. 258).

In Fig. 259 the shaded area represents the regions in which pain is usually felt. Sometimes pain is felt in the same region on the right side, sometimes in the chest behind, in the region of distribution of the same nerves. The presence of pain in any part of the area shaded in Fig. 259 should, in the absence of any definite cause, raise the possibility of the pain being cardiac in origin.

The value of pain in prognosis.—In describing the principles of prognosis it was pointed out that danger to life only occurred when the agents of disease attacked the structures which were concerned in maintaining the heart-beat, the respiration, and the integrity of the capillaries. The structures concerned in the production of pain are not included in this category, though they may be associated with them.

In bowel, renal, and biliary colics, the pain may be excruciating, but the pain does not indicate danger to life. If the bowel colic is due to obstruction of the bowel, then the obstruction is the danger and not the pain.

In every case of pain, where a prognosis is wanted, the pain is not the sign to be sought for, but the condition with which the pain is associated. Pain resulting from contraction of the bowel leads to the inquiry into the circumstances that cause the contraction in the particular instances. So when we come to deal with a case of angina the prognosis is not based upon the pain, but upon the condition which may provoke the pain.

The significance of this lies in the fact that the severity of the pain is not of prognostic significance. Many people who suffer from the most agonizing

pain in attacks of angina pectoris are in no danger of their lives. Sufferers from the secondary anginas never die of their heart trouble, although the pain may be excruciating. Many people with the primary form, when there are

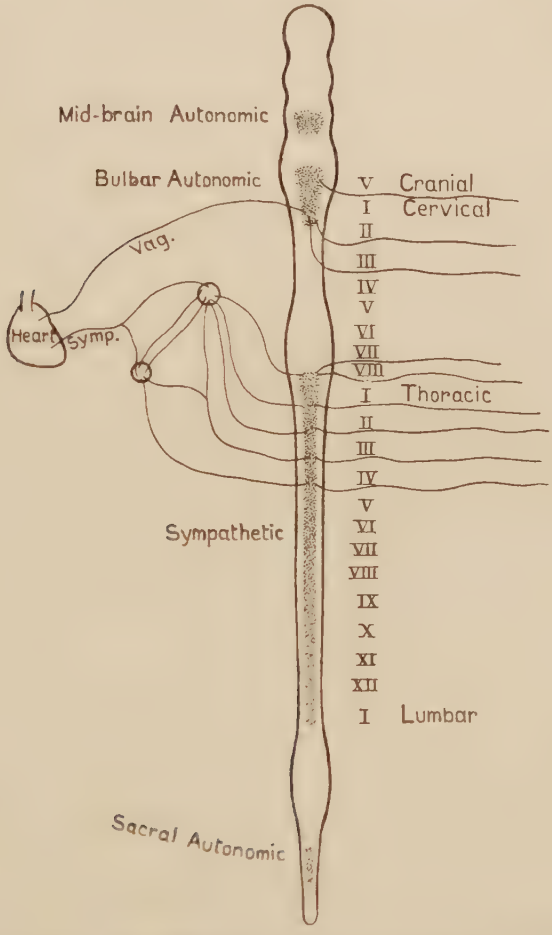


FIG. 258. Diagram showing the different parts of the autonomic or vegetative system in the central nervous system, with the relation of the cardiac nerves to the sensory nerves of the cerebro-spinal nervous system. The vagus conveys impulses from the heart which may affect the 3rd branch of the 5th cranial (giving rise to pain along the jaw), and the 2nd and 3rd cervical nerves (giving rise to pain at the back of the head and neck). The sympathetic nerve of the heart is in close relation to and may affect the 8th cervical and 1st, 2nd, 3rd, and 4th thoracic nerves (giving rise to pain in the chest and arm). (See Fig. 259 (*a*) and (*b*).)

progressive changes in the heart, may lead useful lives for many years after suffering from attacks involving very severe pain. Even in the gravest form of angina the pain may be slight, and when death comes it is seldom immediately preceded by pain.

Angina pectoris and the principle of the reflex arc.—A conception which has guided the investigation of symptoms described in Chapter XIII. is based upon the principle that symptoms are reactions produced by an increase or decrease of activity or a disorderly activity of the organ which exhibits them.

Pain is manifestly due to an increase in the activity of certain portions of the central nervous system.

An increased activity is due to an increase in the number of impulses which stimulate the cells which manifest the activity. It has been shown in the description of stimulation, p. 97, that this process is due to an increased

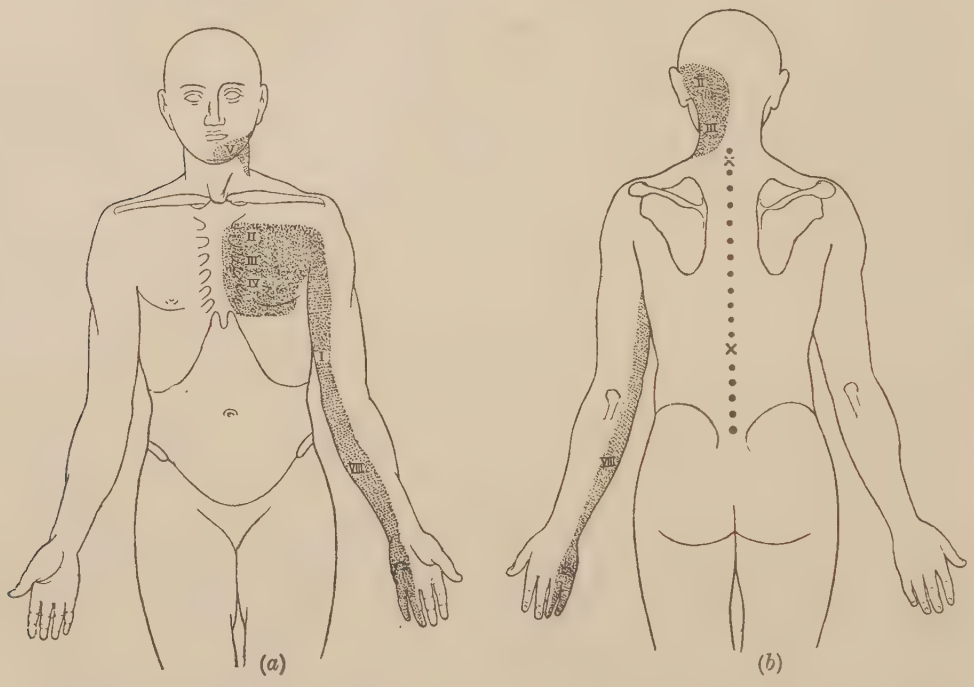


FIG. 259. The shaded areas show the region in which pain is frequently felt in attacks of angina pectoris—along the jaw and back of the head and neck and in the chest and arm. These regions are those in which the sensory nerves are distributed, which lie in close relationship in the central nervous system to the cardiac nerves as shown in Fig. 258.

number of impulses generated and conducted to the functioning cells, or due to an increased excitability of these cells.

Angina pectoris is produced by both these factors.

It has been shown that the latter method occurs in a condition described as secondary angina, where a certain portion of the central nervous system was rendered so excitable that the impulses from the normal heart produced that increased activity that resulted in the pain of angina pectoris.

It has been abundantly shown that the contraction of muscle throws out such a greatly increased number of impulses (as in the uterine contraction of

labour pain) which has the same result in stimulating to activity the cells in the central nervous system which produce pain and other reactions.

Although one can thus trace angina pectoris to the heart muscle, and although we can recognize from the various illustrations given that the voluntary muscles made to contract when deprived of blood give rise to pain, and although it can be demonstrated in many cases of angina pectoris that the heart muscle was deprived of blood, and that pain resulted when there was a demand for more blood, as on effort, yet it is not quite clear how these impulses which could not arise from a greatly increased number of muscle cells contracting gave rise to pain. The matter is left there for further investigation.

CHAPTER LI

PRIMARY ANGINA PECTORIS

The oncoming of angina pectoris.
Attacks of angina pectoris.
The compression of the chest.
The sense of depression.
Vaso-motor disturbances.
Increased flow of saliva.
Vomiting.
Gaseous eructation.
Increased flow of urine.
Manner of death.

The oncoming of angina pectoris. — There are so many circumstances which favour the production of angina pectoris that it is not possible to give a description that would be sufficiently comprehensive. If, however, we recognize the salient features and the circumstances in which some of the more common forms arise, we obtain a knowledge which can be applied to the understanding of most forms of angina. For that purpose it would be advisable to take that form of the complaint which comes on in people during advancing years. This is the most common form, and it is also one which affords the best opportunities of studying angina pectoris in many of its phases, and of correlating the phenomena during life with the morbid conditions found post-mortem.

As years advance people become conscious of their inability to undertake the amount of effort which they used to do in their days of vigorous manhood. Usually the reasons for their limitations are not clear, but not infrequently between 50 and 60 years of age they begin to be conscious of a slight tightness across the upper part of the chest on going up a hill or on running. This sensation has been experienced before, coming on, it may be, towards the end of a race in their school days. By and by, a more distressful feeling becomes apparent, sometimes amounting to pain. At first it is disregarded, or attributed to indigestion, particularly when it is found to occur on walking after a meal. This belief is confirmed because of its disappearance after the employment of some stomachic remedy. Gradually and almost imperceptibly, the limitations increase and the pain becomes more severe and more easily provoked. There are periods when it may seem to have completely gone, followed by periods when it becomes again more readily produced.

PLATE II.

Interior of the left ventricle and aorta.

The cut surface of the wall of the ventricle shows patches of fibrosis on the right side.

The lumen of the coronary artery is greatly diminished.

The aorta is affected only at its base, and nodular thickening of its wall is shown, involving the mouth of the left coronary artery, narrowing it and rendering it rigid and inextensible.

During these latter periods some unusual circumstances, such as an extra effort or a prolonged period of rather more active effort, or exposure to cold, or loss of sleep may occur, and a violent attack of pain may ensue. This pain may last for a few seconds or for many minutes, and even hours, varying in intensity. There may be but one attack, or attack may succeed attack, occurring many times in a day and continuing for many weeks. Usually the attacks gradually subside and the patient may be free from further attacks for periods of varying duration, sometimes for many years. But ever afterwards the patient recognizes the possibility of their recurrence, for circumstances occur from time to time which induce a warning pain.

As time goes on a stage is reached in which the pain becomes easily provoked from a great variety of circumstances, such as effort, exposure to cold, mental excitement, digestive disorders, etc. The patient at the same time becomes more feeble and gradually his strength fails, until he may die from gradual exhaustion with or without symptoms of heart failure, or death comes suddenly and unexpectedly, not necessarily during an attack of angina. He may be carried off with incidental affections with which his failing strength cannot cope.

Such is the picture of this condition which one frequently meets, and it includes most forms of the complaint, the only variations being the rate at which the condition develops and the age at which it occurs.

This picture is typical of patients in advanced years with slowly advancing degenerative changes in the capillaries and arteries of the heart (such as are represented in Plates I.-IV.). In diseased conditions, as distinct from these senile changes, the symptoms arise earlier in life and proceed at a more rapid pace. Speaking generally, the younger in life, before 50, these symptoms appear, the more rapid is the progress. It occasionally happens, as in rheumatic and syphilitic affections of the heart, that the disease advances only a certain length and then does not progress, or progresses very slowly, so that patients may live for many years, suffering, it may be, considerable pain at more or less frequent intervals. Patients in whom the disease behaves in this manner are chiefly those where the rheumatic or syphilitic infection attacks the aorta, usually also the aortic valves. The sclerotic process which these affections produce embraces the mouths of the coronary arteries, reducing their lumen and rendering them rigid, so that it is impossible for them to dilate when more blood is required by the heart muscle during effort.

Pains occur usually in two different ways: they may be produced in response to effort, coming on gradually, beginning with a sense of discomfort across the upper part of the chest, increasing in intensity when effort is continued and ceasing at once when the effort is discontinued. A man may set out to walk and be pulled up after a few hundred yards, and then go on for many miles without any discomfort. Or he may be pulled up in this way several times, and then be able to continue walking without any trouble.

A stage is reached when pain is always provoked, even when distances of only a few hundred yards are covered at a slow pace. There may be no

other forms of pain but this. I saw a lady of 80 years of age not long ago who never had an attack of angina, but she was forced to keep her bed because such effort as walking across the bedroom floor would induce discomfort and pain in the chest. She ultimately died without ever having had a severe attack of pain.

Other circumstances provoke pain which it is difficult to explain. The attacks come on without any warning, although they can frequently be traced to, or be associated with, some definite circumstances. Thus a frequent accompaniment is a sense of chilliness, the hands go cold, and at the same time pain may be of an agonizing kind and come on when the patient is resting. This may occur shortly after a meal. The curious thing about this association of chilliness is its relation to cold. In one patient the attacks of pain were readily produced on getting into bed when the sheets were cold. This induced the sense of chilliness and shivering accompanied by the pain. If the bed were previously warmed, there was no attack of pain. This patient could take a cold bath and enjoy it, and feel a warm glow when he was dried and clothed, but shortly after this glow a reaction in the opposite sense would set in, he would feel chilled, and the pain come on with great severity. He tried a warm bath and enjoyed it, but after he was dressed his hands became cold, and although he got into bed with hot bottles, there followed the most violent attack of pain that he had suffered. This patient could motor on a cold day and become very cold and yet suffer no pain, whereas walking in the cold readily produced the pain, which, however, ceased as soon as he stopped.

I have sought for an explanation of these attacks of pain, which have received the name 'vaso-motor angina', and have discussed the matter with others, and I have taken numerous observations of the blood pressure, of the pulse-rate, and of the rhythm, but although a great many hypotheses have been put forward, there is none which, to my mind, has proved satisfactory. It may be taken for granted that, until we have a better knowledge of the vital processes concerned in these reactions, all hypotheses will be but guesses, and almost to a certainty wrong guesses.

One curious incident was observed when these observations were made. A certain patient's powers of walking had been limited for many years by aching in the legs and feet. On several occasions, after walking a considerable distance in spite of the discomfort in his legs, when resting on a couch some hours afterwards, he was seized by attacks of pain, sometimes of great severity, each attack lasting for about a minute, and felt in the calf of one or other leg. These attacks never occurred when he ceased walking long distances on account of being pulled up by the pain in the chest.

Attacks of angina pectoris.—The onset of an attack, whether in response to effort or when the patient is at rest and from unrecognized causes, begins with an indefinite sense of distress in the chest, not altogether painful, but of such a nature as to make the patient aware that it can get worse. If walking, this sensation may pass off on standing still. If it occurs when resting, it tends to increase in severity, and becomes so severe that the patient writhes in

Interior of the left ventricle and aorta.

At the base of the aorta is a large thickened plate of tissue, calcified in places (brown), involving the mouth of the right coronary artery. The remainder of the aorta is normal.

The cusps of the aortic valve show great nodular thickening.

In the ventricular wall on the right side near the base is a section of a cylinder of degenerated and calcified tissue.

Below the cusp of the aortic valve is the membranous part of the septum, and at its junction with the muscular part of the septum is the left limb of the A-V bundle, which was invaded by the extension of the calcified cylinder.

agony, and tries to get into a position that may give ease—sometimes doubling himself up, sometimes kneeling at the bedside, sometimes standing up and holding to a chair or table for support. The attack may reach a climax and gradually pass off completely, or keep recurring for a few times. Frequently these recurring attacks are not so severe, and they may last longer, keeping him in continuous distress, fluctuating in severity. During the attack various other phenomena may appear. The pain may be felt in the chest or it may spread to the arms or to the head and neck (see Fig. 259).

The compression of the chest.—A frequent accompaniment of the attacks of pain is a sense of compression or of constriction or weight over the chest. Indeed, this sense of compression may cause more distress and alarm to the patient than the pain. A pain is sometimes spoken of as a ' constricting ' pain, which, like a ' sickly ' pain, is a combination of two distinct sensations ; a sickly pain being a pain associated with nausea, while a constricting pain is a pain associated with a sense of constriction. These two sensations are quite distinct, and may occur, the one without the other. Thus, in a man who suffered from an attack of angina pectoris there was no sensation of constriction at first, but after a time the constriction came on with such severity that it caused him more suffering than the pain. In some people attacks of the same nature as angina pectoris may occur without pain, but there is that sense of constriction so compelling that the individual has to stop what he is doing, just as he does when severe pain is present. This constriction is usually associated with a sense of depression.

In seeking for an explanation of the sense of constriction or compression I lean to the view that it is due to a contraction of the intercostal muscles. It resembles in a remarkable manner an attack of pleurodynia, which is a spasm of the intercostal muscles. Moreover, we have also the experiences of pains arising in the abdominal cavity in the different colics, where an attack of pain is often associated with contraction of the abdominal muscles.

The sense of depression.—Accompanying the attack of pain there is often a great sense of depression or oppression, and the peculiar sensation as if death were impending. This is sometimes spoken of as a sense of dissolution. The sensation is not limited to attacks of angina pectoris, because it is found in other conditions, as in attacks of auricular flutter, renal colic, etc. It may also occur in rare forms of migraine, when a wave of extreme exhaustion of the same disagreeable kind passes over the patient. I am disposed to think that this condition may be associated with a depression of the heart's action, and in a few cases, where I have seen it present during an attack of pain, the pulse has become small and feeble. Many people have very severe attacks of anginal pain without this depression, and in some of these I have taken records of the pulse, and have found no alteration either in the character of the pulse-beat or in the blood-pressure.

Vaso-motor disturbances.—In most patients the attack comes and goes with no evident disturbance of the circulatory system, but in some there is marked vaso-motor disturbance ; some patients become pale, others flushed,

and in both instances the attack is followed by a perspiration—sometimes profuse—breaking out on the forehead or over the body. Reference has been made to the sense of chilliness with cold hands.

Increased flow of saliva.—In some cases during the attack of angina pectoris an abundant flow of saliva takes place into the mouth. This is sometimes present when the pain is along the jaw, but also in many cases when the pain is confined to the region of the chest. The stimulus here is probably conveyed to the nerve by the vagus, but which nerve to the gland is stimulated I cannot say. The saliva is sometimes described by the patient as being 'salty' in taste.

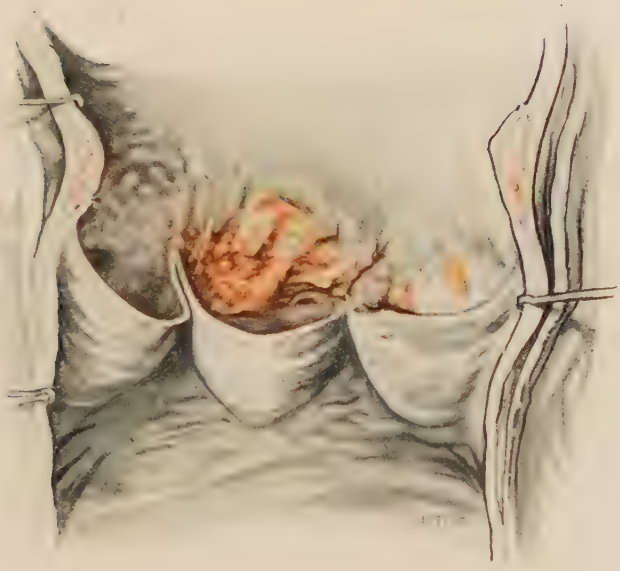
Vomiting.—I have had some difficulty in satisfying myself as to the relation of vomiting to angina pectoris. I have records of patients who said they vomited while the attack was on, but in most the vomiting was probably the cause of the attack. There is generally a history of gastric disturbance when the vomiting came on and the pain with it. In secondary angina vomiting may occur where there is a generalized disturbance, so that all organs are easily stimulated, as I have already described. In one patient the mouth suddenly filled with saliva, and on three occasions this was followed by violent retching. Considering the fact that a flow of saliva is frequently a precursor of vomiting, it is likely that the central nervous system was so hypersensitive that the stimulus passed readily from the nerves that regulate salivation to those concerned in vomiting.

Gaseous eructation.—Many patients are conscious that with the expulsion of gas from the stomach relief is at once experienced. In watching a few patients during the attack, I observed they were attempting to swallow air, and after a time they expelled a quantity with a loud noise, and at once felt relief. This occurrence leads many patients (and some doctors) to the view that the attacks are really gastric in origin, because they are not conscious that they swallow air. In one elderly lady who suffered from a series of attacks, she loudly expelled the swallowed air during the attack. When she was able to walk out, she was quite comfortable on the level, but as soon as she walked up a hill she began to swallow air, and expelled it with such a loud noise that her daughter refused to walk with her. The patient admitted that as soon as she felt the pain she swallowed the air, as its expulsion seemed to afford her relief.

Increased flow of urine.—After an attack has subsided, some patients feel called upon to pass urine, and pass an abundant flow. One patient who had an attack on his way to consult me wished to micturate on entering my consulting room, and he passed a large quantity—pale and of low specific gravity.

Manner of death.—In watching these patients till they die, except where there are intercurrent affections such as apoplexy, disease of the kidneys, gangrene of the legs, the onset of auricular fibrillation, or the occurrence of other forms of disease as pneumonia or cancer, the disease is steadily progressive. The patient becomes weaker and weaker, till he gradually sinks and dies from exhaustion, sometimes suffering little or no pain, sometimes after

PLATE IV.



Base of the aorta opened from the left side.

On the surface and on the sections of the wall are shown patches of degeneration and calcification.

Above the central cusp of the aortic valve is a thin nodular plate which was completely calcified, and narrowed the mouth of the right coronary artery.

The more distal portion of the aorta is unaffected.

a period of great suffering. Occasionally the weakness of the heart muscle becomes such that the output is so diminished that we get, in addition, the evidences of heart failure produced by the deficient output, namely, breathlessness, dropsy, œdema of the lungs, etc.

More than one half of the patients die suddenly, due in all probability, to the occurrence of ventricular fibrillation. This happens frequently when the patient is at rest, being found dead in bed, or when sitting in a chair. It is a painless end, and rarely happens during an attack of angina pectoris, and it should be noted that the death is not due to the condition which provokes the symptoms of angina pectoris, but to the onset of an abnormal rhythm. We would not consider the onset of auricular fibrillation as due to angina pectoris, and the onset of ventricular fibrillation should not be differently regarded.

CHAPTER LII

Angina Pectoris (*continued*)

Prognosis.
Treatment.
Treatment for attacks of pain.
Treatment for the prevention of attacks of pain.
The surgical treatment of angina pectoris.

Prognosis.—It is unfortunate that the human mind is fascinated by the tragic, and those instances, relatively rare, where death speedily supervenes on an attack of angina pectoris, impress the mind and afford an opportunity for graphic or lurid description that few can resist. For this reason angina pectoris has come to be looked upon as of such gravity that there is but one end, and that a speedy one. To relieve the sufferer from anxiety the doctor has had to resort to the comforting description of a form of suffering which is called pseudo-angina pectoris. While we cannot but deplore the inexactitude of medical terms, this term exceeds all others in its futility and absurdity. Pain, being a phenomenon in nature, cannot under any circumstances be false. The employment of the term does grievous harm in that it prevents the doctor from recognizing the nature of the complaint, and therefore missing the opportunities which would enable the patient to take steps to avoid the real dangers which lurk behind his sufferings.

There is a wonderful sameness in the progress of angina pectoris. After the first series of attacks, or even without the severe attacks when there has only been slight distress on exertion, the tendency is for the heart's response to effort to become more and more restricted. The difference in individual cases lies in the rate at which this limitation progresses, and by this rate we have an indication of the rate of progress of the morbid condition in the heart itself. In a measure, this rate can be expedited or shortened by the behaviour of the individual. The systematic avoidance of the conditions that provoke the attack does tend to prolong the life of the individual. This can be partly inferred from the manner of death. My statistics show that more than half the cases of angina pectoris die suddenly, and rarely during an attack of angina pectoris. The tendency is for the ventricles to pass into fibrillation, and there is reason to suppose that increased bodily effort tends to favour the oncoming of ventricular fibrillation in people with degenerative

changes in the heart muscle, as it does in auricular fibrillation. Seeing that the progress of the disease is the ground upon which a prognosis should be based, the information necessary for this purpose is obtained by noting the rate at which the limitation proceeds. It is, therefore, inadvisable to give a final prognosis on first seeing the patient unless we get a clear history of the onset and progress up till the time he was seen.

Further, the prognosis in an individual is also dependent upon the disease which provokes the limitation. If it be of a rapidly progressing kind naturally the prognosis is graver than when it is slowly progressive. Where the angina is due to the slowly advancing changes which accompany old age, the prognosis in people over 60 is relatively less grave. Indeed, the greater proportion of individuals who suffer from angina live for many years after its onset. On the other hand, its occurrence in early life is almost a sure indication that the disease which provokes it is more rapidly progressive, so that death is apt to occur within a few years of its onset.

There are, however, exceptions to this. We may find cases in which the disease has gone a certain length and then ceased, but the patient may suffer for a great many years from attacks of pain on effort. When this happens, particularly in people who have a disease of the aortic valves after rheumatic fever, and even after syphilis, the damage may not be progressive, or very slightly so, and the individual may live for a great many years. Some people with these damaged aortic valves live for a long period, and even lead active lives and suffer little pain, while others suffer great pain. The reason for this has already been suggested, that the diseased process has extended from the valves and embraced the mouths of the coronary arteries, so that they have become stenosed, and on this account the heart does not get that greatly increased supply of blood which is necessary for its work when called upon by bodily effort. In giving a prognosis in heart affections it is always well to remember that the symptoms of heart failure, whether breathlessness or pain, may be extremely prominent in the comparatively early stages. As has been already described, patients tend to ignore the earlier and milder symptoms, and when the symptoms culminate in somewhat violent attacks of pain they create a great deal of alarm in the minds of patients and patients' friends. This alarm is often aggravated by the doctor sharing in it. There is no danger in these early and violent attacks, except in the extremely rare cases where there is some sudden event, such as the blocking of a coronary artery. Patients rarely die in the first series of attacks of angina. I know this is contrary to the widely held beliefs. Some months ago a doctor reported to me the death of a patient in his first attack. I asked him if the patient had not consulted him for other troubles, and he said he had been under treatment for several years for indigestion. On inquiry, I found what he called indigestion was pain in the lower part of the chest on walking after food,—a diagnostic sign of angina pectoris. Some of the most severe sufferings from angina pectoris I have witnessed have been in patients in the early stages of the trouble, and they have lived long periods of years thereafter,

and so long as they avoided the circumstances that provoked the attack they got on very well.

Evidences of gross changes in the heart and aorta do not add much to the gravity of the condition.

OBSERVATION 75.—I saw once a patient whom I had seen about ten years before in London, then about 60 years of age, complaining of slight cardiac distress. Five years previously he had been examined by a London cardiologist who warned him of the gravity of his condition. He wanted to come and see me but was forbidden, as the risk of travelling so far was too great. Nevertheless, he came, and I found he was suffering from attacks of angina easily provoked. The heart was considerably increased in size, and there was great dilatation of the aorta. I told him to carry on, and walk about as much as he could, stopping on the first signs of distress. He again came to see me in 1901, and stated that he had been kept in the house for six months, because his doctor had taken him to another consultant, who, finding a dilated aorta, frightened the patient by telling him that this was a sign of great gravity. I told him to carry on and never mind his dilated aorta, for nobody ever dies from a dilated aorta (see p. 389). In 1904 he was still carrying on, although his field of response to effort was greatly restricted.

It must always be remembered that no one can foretell the oncoming of ventricular fibrillation, and it is foolish to frighten people with a prospect of sudden death when you cannot be sure either that it will come, or when it will come, or the circumstances which would favour its oncoming.

Treatment.—There are few complaints for which so many kinds of treatment have been employed, and every form of treatment finds support in the citation of cases in which it has done good. Drugs of opposite effects are supposed to have beneficial results, great varieties of remedial measures, baths, exercises, massage, diets, and others each and all, are supposed to have been found useful.

In the description given of the morbid condition underlying angina pectoris, and the course pursued by the disease, it will be seen that none of these different remedies can by any possibility have any effect in combating the disease itself, except it may be in the syphilitic cases. The changes that occur in the heart are the same as those which occur in the skin and in the arteries of the legs, and we know how useless remedies are to increase the vascularity of the attenuated skin or to restore the degenerated artery. As the processes which produce angina pectoris are fundamentally of the same nature, the futility of these procedures becomes apparent. The reason for their supposed success is simply due to the fact that the remedy is always accompanied by a great diminution in bodily effort, and in place of attributing the improvement that takes place to the rest, the improvement is attributed to the virtues of the remedy.

This reasonable explanation is rarely acceptable. To both doctors and patient angina pectoris is a mysterious complaint and requires a mysterious remedy. I have repeatedly explained the position at great length to very intelligent doctors who had consulted me for the trouble in themselves.

When they have left I hear from them, telling me of the wonderful improvement they are about to obtain by some fantastic remedy. Sometimes they write to me and tell me of what great improvements they have experienced,

and in reply I ask them to inform me in the course of a year the outcome of their treatment. Invariably there comes back the reply that they are no better, though some say they have discovered another new remedy which is about to cure them of their complaint.

While writing my book on angina pectoris, I communicated with several hundred doctors for the history of the patients who had consulted me. No two patients had followed the same lines of treatment, excepting the comparatively few who had accepted my prognosis and suggestions for treatment, but all the others had fulfilled the prediction that had been given, and in not a single case could a ' cure ' be said to have been found. I wish, therefore, to impress upon medical men the consideration of angina pectoris from the point of view that I have given, that it is the expression of actual changes occurring in the heart (such as those in Plates I.–IV.), and that these changes are as irresponsive to any form of treatment as other changes which accompany old age or diseased conditions which are perceptible in other organs.

If, then, they recognize that there is nothing mysterious about the morbid condition which gives rise to angina pectoris, but that it is due to irremediable changes such as occur in other places, as, for instance, the arteries of the leg, and that the limitations imposed by the heart and the symptoms which arise are provoked in a manner similar to that which occurs in the leg, we find that the principles which guide treatment are also the same. In both instances, exercise beyond a certain limit produces suffering. If persisted in in spite of these evidences of exhaustion, the suffering will become increased and prolonged.

In the management of individuals with angina pectoris it is advisable to explain to them the nature of their limitations, because, unless a man recognizes what these are, it is impossible for him to lead a life that will make the best of his powers, however limited, and to avoid those circumstances which tend to increase his distress and hasten the exhaustion of the heart muscle.

It is the prevention of this exhaustion to which all efforts should be devoted, and in this respect, the suffering of angina pectoris is not without its compensations. Taking the pain as a symptom produced by exhaustion, its imperious character acts as a restraining influence. If the patient will carefully observe the circumstances that provoke the pain he can take steps to avoid them. Thus, in walking, if he becomes conscious of a slight feeling of distress across the chest it may be experience tells him that if he continues his effort the pain would increase until he was compelled to stop. If, then, he slows down and stops on the first appearance of the slight distress, and does not resume his walking until it is completely gone, he may go on for years and never suffer from any pain.

Many patients find, even in the early stages, that a great many years before the condition becomes serious, walking out after a full meal gives rise to pain. This point is a very important one, for I find some writers declare that that is a sign not of heart trouble, but of indigestion. If, however, the site in which the pain is felt be carefully examined it will be found to

occupy some portion of the region shaded in Fig. 259. Moreover, the cessation of the pain when the effort is stopped is a distinct indication of its cardiac origin.

Such patients should take meals small in quantity, and should rest for half an hour or so after each before they attempt bodily effort. Such people may be capable of undertaking a considerable amount of effort without distress, walking long distances, climbing hills, and engaging in laborious occupations for a long time after the pain has appeared. Thus a workman, such as a stone-mason, may have difficulty in reaching the scene of his labours on setting out after breakfast, but he may carry on his work all day with no distress.

Another provoking cause is exposure to cold. Quite a number of people in the early stages only feel the pain when walking in the cold air or against a cold wind. Some only experience it if they have to go out in the night. Such people, again, may lead laborious lives for many years after the onset of pain provoked during exposure to cold. Some people in the early stages may have extraordinarily violent attacks of pain when exposed to cold alone, as in a railway carriage. The pain may persist when the body has become quite warm.

As the condition gets worse, this susceptibility to cold may increase, so that pain may come on in passing from a hot room to a cold, and many experience pain on first going to bed because of the cold sheets. Recognizing this, care should be taken to avoid the circumstances which the patient recognizes provoke the attack. By means of warm clothing and warm bed clothes and slight exertion in the cold, such attacks may be completely avoided.

These are given to indicate some of the more common circumstances which give rise to the pain. In individuals a variety of other circumstances may predispose to the pain, and these also have to be carefully scrutinized to know how they are to be avoided.

Treatment for attacks of pain.—When the pain is on it is necessary to resort to measures that will give relief when it does not subside with the cessation of effort, or when it occurs and persists in circumstances where no effort is made. For this purpose the remedies which cause dilatation of the peripheral vessels are frequently very efficacious. Such remedies as brandy or whisky, if possible in hot water, are often helpful. Nitro-glycerine or trinitrin, 100th of a grain in tablets, is often successful in relieving the distress. In the use of these latter remedies they should be given every four or five minutes till relief is obtained or until 4 or 5 have been taken. If after this there is no relief other remedies must be resorted to. Nitrite of amyl acts in a similar way, but more speedily and more certainly than any other drug. It is usually supplied in capsules containing 3 or 5 drops. These capsules are broken, and if the patient is careful to inhale fully the drug, one capsule is usually sufficient to give relief. If not, a second may be tried, but if the inhalation is carried out properly, more than this is useless if relief be not obtained.

When the pain persists in spite of such remedies, or keeps recurring at

frequent intervals, hypodermic injections of morphine are the best of all remedies. If the pain be very severe, ½ a grain should be injected, and if no relief is obtained, injections of ¼ of a grain should be continued every quarter of an hour until the patient is brought under the influence of the drug. In some cases where the pain is very severe and keeps recurring at short intervals, it may be necessary to repeat these injections for several days.

Where the pain is of an agonizing description and the morphine is long in taking effect, chloroform may be required to produce unconsciousness for five or ten minutes until the morphine has time to act.

Treatment for the prevention of attacks of pain.—In some cases a stage is reached when the tendency to attacks is so great that there are times when the slightest effort, such as walking a short distance, having the bowels moved, or even taking food, may induce an attack of pain. Also mental effort, such as a discussion of an object of deep interest, may have the same effect. Before undertaking any of these efforts, a small dose of nitro-glycerine may enable the effort to be performed with comfort. A handy form of the drug is the hypodermic tablets (1/100 grain) by Parke, Davis & Co.—not used hypodermically, but swallowed. The effect begins to be felt by a fullness in the head within a minute or minute and a half, even when taken after a meal, and the effort can then be performed in comfort.

The surgical treatment of angina pectoris.—There has, in recent years, been an endeavour on the part of surgeons to seek new fields for operation, and there seems to be no region of the body which the surgeon is not prepared to invade. The impression is that the feats of the surgeon are indications of the progress of medicine. When operations are carried out with a full knowledge of the morbid conditions which it is intended to relieve, and with a knowledge of the functions of the structures with which the surgeon deals in his operation, there might be reasons for regarding such surgery as an example of the progress of medicine, but seeing that in many instances the surgeon is profoundly ignorant of the morbid condition for which he operates, and of the functions of the structures which he mutilates, it is impossible to conceive anything more detrimental to the progress of medicine. At one time the surgeon was supposed to have a knowledge which would enable him to make not only a diagnosis, but a prognosis, so that he could tell whether his interference would be to the patient's benefit. But the trend of modern medicine with its specialism is, that the surgeon dispenses with this kind of knowledge and relies upon others for instructions when to operate. He is thus tending to become more and more merely a dexterous user of the knife and the needle.

This tendency is well illustrated in the surgeon's excursions into the treatment of angina pectoris. I do not suppose there is a surgeon who would even pretend to understand the morbid condition which gives rise to the symptom complex called angina pectoris. The knowledge of the functions of the vagus and the sympathetic which the surgeon cuts is so imperfect

that neither he nor the physician who advises him understands more than a fraction of the functions of these nerves.

At this time of day the subject of angina pectoris is so little known that it is only a very ignorant man who would have the courage to recommend the operations that have been undertaken. While I am far from saying that the description of angina pectoris given in this book is the last word on the subject, yet the description given is sufficient to reveal how foolhardy a procedure is any surgical operation for this condition.

There are a great many people in whom there is no disease of the heart, who suffer from 'secondary' angina pectoris. The suffering in these cases may be extreme, but the patient is in no danger from his heart, the heart condition being secondary to morbid processes elsewhere.

There is another great group of individuals with progressive degenerative changes in the heart who suffer from angina pectoris (primary angina pectoris). In them the morbid condition is of the same nature as that which accompanies advancing years, the progress is slow, but inevitable. These folks for long years are in no danger of death. The pain they suffer is protective. One of the great dangers in heart affections is the systematic overworking of the heart—either because there are no symptoms of distress, or because these are not understood. Although pain may not be considered beneficent, in some respects it is life-saving, for it calls attention to conditions that, if ignored, will imperil, not only the health, but the life of the individual. In many people it indicates the limits of the exertion which the patient may undertake with safety. In the vast majority of those who suffer from angina pectoris the pain is of that nature. No one of any intelligence would suggest that the cutting of a nerve which conveys impulses to the brain would improve the morbid condition which gives rise to these impulses. Beyond the relief of pain, this operation cannot benefit the patient by improving his condition, or delaying the progress of the disease. But in removing the indications of exhaustion, it is bound to favour exhaustion, which is one of the dangers to be guarded against.

The disease which gives rise to angina pectoris may be so advanced that the individual's life is in constant peril, and to undertake an operation such as is proposed and carried out cannot do other than hasten death. Let any one look at the damaged heart muscle or the blocked coronary artery in Plate I., which are so frequently found in people who have suffered from angina pectoris, and he will see that the cutting of the vagus and sympathetic nerve can never help these morbid conditions.

In taking up this attitude I will be accused, as I am often accused, of being a reactionary, and of trying to obstruct the triumphal progress of surgery, but where such operations are recommended for conditions so little known, great harm is done to the individual, while medicine is brought into discredit, and its progress obstructed.

I have taken the opportunity of studying carefully such cases of operation for angina pectoris as I have found recorded. I find no attempt to

differentiate the condition on the lines I have just indicated, consequently I find in the records individuals operated upon who had no disease of the heart, the angina being manifestly of the secondary type, others being operated upon who were in no danger but who got relief from their suffering, but there was no record of the effect of the operation on their future. Finally, there were patients operated upon who had undoubtedly got advanced disease of the heart, and they died—their death being directly caused by the operation.

In expressing my views strongly upon this subject, it is not only because such operations inflict grievous injuries on the patient, but because the attitude of mind which lies behind these operations is one of the greatest dangers to medical progress. Hitherto, medicine has been made the object of ridicule and satire on the ground that remedies, whose action is unknown, have been given for conditions of which the doctor was ignorant. Fortunately, such remedies usually do no harm, even when they do no good, but when it comes to a surgical operation, where structures essential to health, and it may be to life, are irreparably damaged, it is necessary to draw attention to a course of action which is, to say the least, a step backwards.

CHAPTER LIII

Angina Pectoris (*continued*)

Secondary angina.
The significance of the secondary form of angina.
Prognosis.
Treatment.

Secondary angina.—Reference has been made to the fact that pain may arise from the great increase in the number of impulses, or from an increased excitability of the nerve cells in the central nervous system concerned in the production of pain. The former accounts for the pain of angina pectoris due to disease of the heart, the latter accounts for the pain of angina pectoris when there is no disease of the heart.

There is no difference in the attacks. The pain comes on in secondary angina in the same way as it does in primary, except that attacks apart from exertion and such sensations as chilliness and shivering are more common.

The most distinctive features of secondary angina are the age and sex of the individual. It is far more common among women, and particularly in women under 55 years of age. Indeed, I have for many years looked upon angina pectoris in women under this age, more particularly when they are younger, as being invariably of this nature, if one can exclude gross organic changes such as disease of the aortic valves and mitral stenosis.

Another distinguishing feature between the primary and secondary angina is the condition of the patient between the attacks. As a rule, however violent the attack of angina pectoris may be in the primary form, as soon as the pain subsides the patient feels well and bright. Indeed, it is a characteristic of all cardiac complaints that as soon as the patient is free from the distress which the heart has induced, the feeling of well-being is unimpaired. On the other hand, in secondary angina patients almost invariably feel sickly or languid, or out of sorts in some way, restless and irritable, and often sleepless.

The reason for this lack of well-being in the secondary angina is that the ill-health is due to some other cause, usually of a toxic nature. As soon as the toxic condition is removed, all cardiac symptoms disappear. This disappearance of violent attacks of angina pectoris is sometimes dramatic in its suddenness.

OBSERVATION 76.—A patient, aged 51, whom I saw in consultation, was having attacks of angina pectoris, fifteen or twenty in a day, although confined to bed. These continued three or four weeks, then one day there was a profuse discharge of pus from the bladder, evidently from the bursting of an abscess. The attacks of angina immediately ceased, and there had been no return of heart attacks when I last heard of her nine years later.

The significance of the secondary form of angina.—Hitherto there has been a tendency to divide angina into true and false or mock. Such distinctions are to be deprecated as they give a false impression. In nature there is no false phenomenon, and the condition which I have called 'secondary', and which would come under the name of pseudo-angina, is just as true and real as is the primary angina. The value of the differentiation put forward here lies in this, that when all the facts are carefully considered and the conclusion arrived at that the condition is primary or secondary, the prognosis and treatment will naturally be influenced by the decision. In the secondary form further investigation will have to be carried out to discover the source of ill-health. It will be seen that the cardiac phenomena, however prominent, are but a part of the phenomena of ill-health, and not the most diagnostic of the phenomena, so that a search has to be made through the whole body for evidences of a disturbance in some other part. The source of disturbance may be found by evidences in other parts of the body. A fairly common source is in the abdomen. The symptoms there complained of by the patient may be slight or absent, nevertheless a careful palpation of the abdomen may reveal areas of tenderness or of rigidity in the abdominal wall. Such signs surely indicate disease processes in some of the abdominal organs.

It may be that no such local symptoms can be detected, in which case one is forced to conclude that the patient is subject to some condition which leads to a general toxæmia, probably from microbic infection. Such conditions were fairly common amongst soldiers during the War. Cardiac pain, amounting at times to attacks of great severity, exactly of the same nature as those in primary angina, were not infrequent among young men. In such cases no abnormal sign could be detected, but the histories indicated an infection, many of them showing a rise of temperature. Their after histories were confirmatory of this view, some making a complete recovery, the nature of the infection never having been recognized; others developing ultimately symptoms of tubercular or malarial infection or trench fever. In the absence of a local disease, of a definite infection, or of menstrual irregularities, many patients are described as suffering from 'neurasthenia',—a term, be it noted, which merely indicates a condition of ill-health whose source and nature are unknown.

Prognosis.—Prognosis of secondary angina is not dependent upon the heart condition but upon the nature of the condition of which the heart symptoms form but a part. However great the suffering may be, that is no guide to prognosis. As I have already remarked, pain is due to disturbances of structures which are not concerned in the maintenance of life. As a matter of fact, I cannot recall a single instance where a patient has died during the

period of suffering from cardiac pain. The vast majority of my patients made a good recovery, and even those whose health was impaired on account of the primary condition, lived for many years, and the cardiac signs disappeared.

Treatment.—While the treatment should be devoted to combating, when possible, the cause of the disease, it may happen that as our efforts are unavailing to discover and get rid of the cause, we must employ methods to give the patient relief. The nitrites may be used as in the treatment of primary angina. As in secondary angina patients are often sleepless, restless, and irritable, the judicious use of sedatives, particularly of the bromides, is often very helpful. Drugs should be given in the same way as directed in dealing with primary angina. Alcohol should be avoided.

CHAPTER LIV

The Heart in Debility and the Soldier's Heart

'Debility.'
Differential diagnosis.

'Debility.'—There are a great number of conditions of ill-health in which heart symptoms are present, and those have received distinctive names at one time and another, such as the 'weak' heart, the 'debilitated' heart, the 'soldier's' heart.

When a man falls ill every organ of the body may be disturbed, and in most illnesses the heart participates in the disturbance. Medical knowledge has not advanced far enough to enable us to recognize the nature of ill-health, except in only a small proportion of the sick. The custom has therefore been followed of attributing the ill-health to the organ whose symptoms strike the observer. We thus find patients going from one learned physician to others, and receiving a different diagnosis of their complaint according to the predilection of the physician. One of the physicians is interested in the blood, the ill-health is due to some form of anæmia; another is interested in the digestion, intestinal toxæmia is the cause of the trouble; and another in bacteriology, when all the fauna at the orifices of the body are studied and the ill-health attributed to the microbe which is rarest or most abundant. If there be a murmur or an irregularity or a little albumin or sugar in the urine, then the heart or the kidney or the pancreas becomes the reputed source of ill-health.

This type of ill-health is common in general practice, and in watching these cases I found that a certain proportion ultimately developed forms of disease which could be recognized, so that the heart symptoms could be referred to the early stages of some disease. Some patients afterwards developed tuberculosis and other chronic forms of disease. A condition of ill-health may follow an acute illness such as influenza, in which the heart symptoms predominate, and the presence of such symptoms may compel a search for the conditions that provoked them. The significance of this statement can be seen from the following two instances.

OBSERVATION 77.—Early in my career I was consulted by a lady whose only complaint was great weakness and breathlessness on exertion, and the only physical abnormality was a persistent increase in the rate of the heart which varied from 90 to 120 beats per minute. Repeated

examinations and a consultation with a distinguished physician failed to throw any light on the condition. After a time, she was seized with a hæmoptysis and died a few weeks later from acute pulmonary tuberculosis.

OBSERVATION 78.—The other case was a girl 15½ years of age, who complained of weakness with breathlessness on slight exertion. The only thing I could detect amiss was a persistent pulse-rate of 130 to 140 beats per minute. I tried many remedies to lessen the rate, pushing digitalis on two occasions till nausea and vomiting occurred. My attempts at treatment were useless. One day she called to see me, and as she walked into my consulting room I observed a very slight stoop and limp as she walked. Asking the reason for this she said it hurt her slightly in the groin when she walked upright. On examination I found a psoas abscess.

When the War broke out a great many soldiers returned from the front suffering from various forms of so-called heart trouble, and within the first year tens of thousands were lying in the hospitals. I was asked by the Medical Research Council to make a report upon a number, and with the assistance of Dr. R. M. Wilson we examined 400 cases. In the report we drew up we pointed out that about 90 per cent at least of the cases said to be cardiac were patients in whom the heart symptoms were merely a portion of the evidences of ill-health, and that in the majority the cause of ill-health could be traced to some infection. We had recognized that what was called ' Soldier's Heart ' corresponded exactly to the conditions we had found in civil life. The symptoms also agreed with the descriptions of the soldier's heart found in other wars, and more particularly with that given by the surgeons Hawthorne and Da Costa in the American Civil War. After this, in this country and other countries, hospitals were set aside purposely for the observation and treatment of these cases, and a great number of people engaged in a research with a definite object to find out the nature of the trouble. If one looks at the post-war writings for any evidence of an addition to our knowledge as a result of this research, there will not be found any fact of the slightest importance which throws new light upon any form of heart trouble.

The purpose of this reference is not to make any reflections upon those who engaged in such a research, but to illustrate the very important point that medical knowledge has not advanced far enough to enable us to know how such a problem should be attacked. I may say that I also engaged in a strenuous attempt to obtain a better insight into the matter, and I had no more success than others. My failure so impressed me that I saw the need of some new concept based upon some sound principle which should guide to the solution of this and other problems connected with ill-health. The desire to obtain this concept was one of the reasons for the starting of a Clinical Institute specially devoted to the search for such a principle.

The object of these remarks is to lead the doctor to recognize that cardiac symptoms in people who are ailing, however prominent they are, may be and often are but a portion of the symptoms of ill-health in any given case. When this is recognized, it behoves the doctor carefully to consider the history of the patient, particularly from the time when he began to recognize that he was not well. In this way the origin of the trouble may be traced to some

seemingly trivial matter, such as a sore throat, a little diarrhœa, a febrile attack, or to unhealthy circumstances and surroundings.

Differential diagnosis.—I spent a good deal of time in trying to find out how one could distinguish an ill-health directly due to heart weakness, from one in which the heart weakness was secondary. In this I found a very simple observation, extremely helpful, viz. by finding the nature of the sensations that calls the patient's attention to his weakness. When a patient, for instance, states that on exertion, as in walking, particularly upon the level, he is pulled up by pain in the chest or breathlessness, then the probabilities are that it is the heart which is producing his limitation. If, on the other hand, he becomes tired and exhausted after walking a relatively short distance, that is, a distance much shorter than he was able to undertake with comfort in good health, then it is always certain that the limitations are due to conditions other than the heart.

The most striking differences between a secondary cardiac symptom and a primary have been referred to more particularly in dealing with angina pectoris. However great the distress the heart produces as dyspnœa or pain, in a few minutes after the distress subsides the patient feels quite well, and as if able to undertake various forms of effort. Such effort as is within his capacity he can perform without any feeling of discomfort. On the other hand, in ill-health, where the cardiac symptoms are but a portion of the disturbances, the origin of ill-health being elsewhere, the attacks of dyspnœa or of pain may be slight or severe, but the patient has not that feeling of well-being, but has a feeling of exhaustion or other vague and indefinite sensations by which he knows that he is not well. The following observation brings out very clearly the difference between primary and secondary cardiac conditions.

OBSERVATION 79.—A young soldier was training for a race, and as was his custom, went one morning to run round the track for an hour, but after a short time he fell down in a state of collapse. He was carried in and put to bed, and the doctors diagnosed a 'strained heart'. I was called to see him and, recognizing the circumstances, I asked him how he felt before he began to run. He said he felt seedy, and he thought the exercise would throw the feeling off. His temperature was taken, and it was found to be slightly raised. It turned out to be the beginning of influenza of a mild kind, and in three weeks' time he resumed his training and ultimately ran his race.

CHAPTER LV

Sounds and Murmurs, Normal and Abnormal

The part played by the valves in producing ill-health.
The significance of murmurs.
The attitude of the profession to abnormal sounds.
Variations in the sounds of the heart.
The cause of functional murmurs.
The quantity of blood thrown back to cause a murmur.
The differentiation of functional and organic murmurs.
The significance of functional murmurs.
The significance of organic murmurs.

The part played by the valves in producing ill-health.—The purpose of the valves is to direct the flow of blood. They are so delicately hinged that they respond to the slightest difference in pressure, so that when the pressure on one side is higher than that of the other, they either open or close. The orifices which they open or close are of a size which has a relation to the cavities on either side, and their size may be taken to be that which is best adapted to the flow of blood.

Like all other structures, those which compose the walls of the orifices and the valves are liable to disease, which may result in changes which embarrass the heart muscle in its work, by offering an obstacle to the flow of blood. Valves are not structures which interfere with any vital process. The significance of this lies in the fact that the effect of valve competence or incompetence is not to be determined by the signs the valve may produce, but by the evidence or absence of evidence of muscular embarrassment. When dealing with the modified sounds of the heart this matter will be further discussed.

The valves play another part in the production of ill-health in that they are frequently the seat of morbid conditions which result not only in damage to the valve but in the production of grave states of ill-health, as they may become the site of infective foci. It is probable that the selection of this site is because of the poor blood supply of the tissues which constitute the valve. There seems to be a great tendency for infective organisms to flourish where the blood supply is smallest, and although the valves are bathed in blood yet the blood only affects their surface.

The significance of murmurs.—Before entering on the study of valve

affections, it is necessary, in order to understand the significance of murmurs, to appreciate the following facts :

1. That murmurs may appear in hearts with no damage to the valves, and in hearts with damaged valves.

2. Individuals may show murmurs and be in perfect health, and lead strenuous lives, and never show the slightest sign of heart failure. From this we can conclude that murmurs may be a physiological and normal event, and indicate neither impairment of the heart's efficiency nor foreshadow the oncoming of heart failure. Murmurs may be present in debilitated individuals, and in these the murmur is not necessarily indicative of disease ; nor is the valve incompetence the cause of the debility.

3. Murmurs may be associated with heart failure of a grievous kind. In many cases it will be found that though the murmur is associated with the heart failure, the valvular incompetence may not be the cause of the heart failure, but may be one of the changes which have arisen in consequence of impaired functional efficiency of the heart muscle.

4. Individuals with organic murmurs — the result of damaged valves — may lead vigorous lives, being engaged in strenuous occupation, and never show any signs of heart failure. From this we can conclude (*a*) that valve lesions of themselves are not necessarily of serious significance, (*b*) that the affection which causes the valve lesion has ceased to progress, (*c*) that little or no embarrassment to the heart results from the damaged valve, and (*d*) that no coincident damage has occurred to the heart muscle, or at least not so marked as to impair its efficiency.

5. That heart murmurs may be due to valve lesions which embarrass the heart and aggravate heart failure.

The attitude of the profession to abnormal sounds.—As a general rule, the more obtrusive a symptom is, the more it impresses the mind of the observer, and much more importance is attached to it than to less conspicuous phenomena. This is particularly noticeable in signs which are detected by auscultation. To the human mind, sounds arising from obscure causes have always been a source of mystery, and the human imagination, when dealing with the mysterious, invariably associates it with something malign. This peculiar trait is shown by the attitude of the profession towards murmurs and abnormal sounds of the heart. Within a few years of the discovery of the stethoscope it was found that people who died from heart failure often exhibited murmurs of different kinds, and hence it occurred to the physician of that day that murmurs were manifestations of diseases of a serious nature. This notion was taught at a period when the cause of the sounds of the heart, and much less the cause of the murmurs, was not understood. As years went on, and investigation into the sounds of the heart ultimately brought out the true origin of some of these, the inquiry into the cause of the different murmurs was diligently pursued, and their origin at the different orifices gradually established. Time after time, physicians were able to indicate at which orifice a murmur was

generated, and confirmation of the predictions was found repeatedly at the post-mortem examination. It came to be looked on by the profession that murmurs were an evidence of disease of the valves. As, however, many cases showed murmurs during life, particularly systolic murmurs, and the valves were found intact at the post-mortem examination, a ' functional ' murmur was recognized. But although these functional murmurs were not considered evidence of disease, they came to be regarded as evidence of impairment of the heart's efficiency, and as such to be indications for treatment and a restriction of the life of the individual.

So deep an impression have murmurs made in the minds of the profession, that a large proportion deems them invariably an evidence of an unhealthy heart ; the result of which is that we find every form of murmur looked on as an evidence of impairment, if not of disease. It is true that many physicians recognize and acknowledge that some functional murmurs may be harmless, but when they attempt in their writings to deal with this problem, their words convey so confused an impression, that it is evident that they have but a hazy conception of the manner in which a harmless functional murmur is to be distinguished from a murmur which may be an indication of, or associated with, heart failure.

Variations in the sounds of the heart. — The first sound is produced by two factors, the contraction of the ventricular muscle and the sudden stretching of the mitral and tricuspid valves. Composed of these two elements, it is subject to many modifications, according to changes in the muscles or valves. Consequently, alteration in the character of the first sound is frequent, and satisfactory explanations for these variations are not forthcoming. Moreover, experience has shown us that little can be made out of these modified sounds alone, and while the modification should be noted, the real opinion of the heart's condition should be based on other evidences, which are never lacking. A certain number of modified first sounds have gained a place in the terminology of cardiac affections, and therefore need a brief reference.

Feebleness or absence of the first sound may indicate extreme exhaustion of the heart muscle, in the presence of other signs of heart exhaustion. A ' flapping ' first sound is sometimes spoken of as indicative of a thin-walled ventricle, but we must be chary of trusting such an explanation.

A reduplication of the first sound may be due to a slight difference in the time of contraction of the two ventricles, or at least to a minute difference in the times of the closure of the mitral and tricuspid valves. Recently it has been found that a reduplicated first sound may indicate damage to the branch of the auriculo-ventricular bundle leading to the right ventricle, thus giving a lack of synchronism in the times of the contraction of the two ventricles.

The second sound is valvular in its production. An accentuation of the aortic sound is present when the aortic pressure is raised, and the pulmonary second sound may be found slightly accentuated when there is engorgement

of the lungs ; its cause is possibly due to increased pressure in the pulmonary artery.

Reduplication of the second sound is usually loudest at the mitral area. It is not clear how it is produced, but it is so often present in mitral stenosis that its detection should always arouse the suspicion of stenosis of the mitral valve in the absence of more reliable evidence.

A curious modification of the sounds of the heart is found when there appears a third sound, interpolated between the first and second sounds. The triple heart sound or gallop rhythm has been the cause of a good deal of controversy. There appear to be different conditions producing it ; one of those is the reduplication of the first sound as already mentioned, due to a lesion of the right branch of the auriculo-ventricular bundle. Some have looked upon it as a grave sign, and no doubt it occurs towards the end of heart failure in such conditions as kidney disease ; but I have found it in individuals who were free from any cardiac lesion, and who have lived for many years with no evidence of heart failure.

Muffled sounds seem to be of little significance ; sometimes they change into murmurs.

The cause of so-called functional murmurs.—There are some murmurs whose nature is so obscure that they are still the subject of discussion. Certain murmurs can, with fair certainty, be referred to definite orifices of the heart. Organic murmurs are due to lesions affecting the valves, and these can often be identified by certain peculiarities of the murmurs. There are, however, murmurs which arise when there is no lesion of the valves ; these are spoken of as functional murmurs, but many of these are really physiological in the sense that they occur in perfectly healthy hearts. The term functional murmurs conveys the idea that they are indicative of some impairment. Such an idea is not justified. These murmurs usually occur during ventricular systole, and may be heard in various regions of the heart. When heard loudest at the apex, they are assumed to be mitral in origin ; when loudest over the middle of the sternum, they are supposed to be tricuspid in origin. These murmurs are not infrequently heard loudest towards the base of the heart, and it is difficult to determine in which particular region, tricuspid, aortic, or pulmonary, they are heard loudest, and it is even suggested they are cardio-pulmonary in origin. It is not possible, in all cases, to distinguish them from organic murmurs. Much study has been devoted towards finding out if the mitral functional murmur could not be distinguished from the organic murmur, because of its character and the regions into which it is propagated. While many organic murmurs do show very characteristic features, others resemble functional and physiological murmurs so closely that it is not always possible to speak with certainty.

Functional murmurs are usually assumed to be due to dilatation of the heart, rendering the orifices so wide that the valves fail to close them. This is not entirely correct, for while it is true that we may find a murmur with dilatation of the heart, we may often find great dilatation and no murmur.

Further, we find murmurs come and go when there is no perceptible change in the size of the heart, the murmurs appearing only when the heart is excited or when the individual stands, and disappearing when the heart quietens or the individual is at rest. Again, murmurs which we find when the heart is acting quietly with the body at rest, may disappear on exertion or excitement.

From this it can be inferred that there is some special mechanism, apart from that involved in the dilatation of auricle and ventricle as a whole, and this obscure mechanism varies under circumstances which we are still unable to appreciate. The importance of this is seen in connexion with the heart of the young, where it is probable that the heart has a peculiar power of adapting itself, which is gradually lost in adult life. It must be remembered also that free regurgitation can take place at the mitral and tricuspid orifices without murmurs. We frequently get evidence of marked tricuspid regurgitation in the veins of the neck and in the liver, with no tricuspid murmur.

The quantity of blood thrown back to cause a murmur. — Seeing that the chief importance attached to regurgitant murmurs is that they are evidences of blood escaping back from the contracting chamber, and that the regurgitation has been assumed to be the chief factor in producing heart failure, according to the back-pressure theory, it is desirable that we should form some idea of the quantity of blood which is actually thrown back. Unfortunately, there are few accurate data upon the subject, and we are forced to estimate the amount by indirect methods. I have been able to keep under observation, for twenty or thirty years, individuals with loud, rasping systolic mitral murmurs, and who gave a history of rheumatic fever, so that I could safely conclude that the murmurs were due to some damage of the mitral valves. These patients never suffered from heart failure ; and I venture the conclusion that, in them, the leak was small and the damage slight, so that one may say, within certain limits, that in regard to mitral murmurs the narrower the orifice for the leaking blood the louder the murmur. Where there are functional murmurs, the leak, if this be present, causing them is also slight, and never such as to embarrass the auricles in their work, apart from cases where there is grave damage done to the heart muscle. The small quantity of blood thrown back does not alter appreciably the size of the auricles, for we often find murmurs in hearts of the normal size. A better method of estimating the quantity of blood thrown back in tricuspid regurgitation is to measure the size of the *v* wave in cases with the auricular form of venous pulse. I have sought for evidences of the amount in a large number of cases with a tricuspid systolic murmur and was unable to find any perceptible increase in the size of the *v* wave. Now if the regurgitation had been extreme, the appearance of this wave would have been earlier in the period of ventricular systole. I have noted its size in individual cases where the tricuspid systolic murmur was present and where it was absent, and could detect no change in the size of the wave.

It may be stated that I was mistaken in thinking the murmur tricuspid in origin, for a great many people are under the impression that tricuspid regurgitation occurs only in the last stage of extreme heart failure—a fallacious notion which is the outcome of the back-pressure theory of heart failure. I have examined cases where the tricuspid valve was damaged, so that tricuspid regurgitation was bound to occur, and even in these cases the *v* wave was no larger than that we find in normal hearts (Fig. 49).

Nor does it appear that in aortic disease the loudness of the diastolic murmur is any measure of the amount of regurgitation. I have watched patients for years with a loud diastolic aortic murmur, and they never showed any trace of heart failure which might be referred to the effects of regurgitation. Moreover, the experimental work of Henderson has shown that a slight damage to the valve, permitting the leakage of only a few drops of blood, will cause a marked diastolic murmur and the collapsing pulse. In aortic regurgitation there is often great dilatation of the smaller arteries, and it is concluded that the collapsing pulse is not entirely due to the regurgitation, but due, in part at least, to some reflex causing a dilatation of the arterioles and capillaries, which takes place when the aorta is damaged.

The differentiation of functional and physiological from organic murmurs.—It is not always easy to distinguish between these murmurs. As a rule, functional and physiological murmurs are systolic in time, and are heard usually at the base, middle of the sternum or apex. They are usually soft and blowing, but these characteristics do not distinguish them from some organic murmurs. Much has been written to determine the different features; but while the character of the organic murmur may often be distinctive, organic murmurs, both in their character and propagation, resemble the others so closely that it is not possible in all cases to differentiate them. A very rough murmur, especially if accompanied by a purring tremor or a musical note, is indicative of a valve lesion. In the early stages, however, of an endocarditis the murmur is usually soft and blowing, resembling a functional murmur. It is necessary to distinguish between these two murmurs, and to do this the whole circumstances of the case must be considered. In the first place, it is necessary to bear in mind that a murmur may be a normal event. When we find a murmur occurring in an individual in robust health, and if we can safely assume it to be physiological, we may consider it as indicative neither of impairment nor disease. On the other hand, when we find a murmur occurring in debilitated persons, search should be made for conditions which account for the debility, as anæmia, imperfect nourishment and rest, or disease of other organs. It is of importance to differentiate these murmurs in febrile cases, or after the subsidence of an acute febrile attack, as after rheumatic fever. The question then arises whether the heart has been affected by the disease process. Here it is necessary to bear in mind that fever alone may produce changes in the heart's action, such as increase in size and murmurs, so that during the febrile stage of such

acute illnesses judgment should be suspended until more definite signs of an organic lesion are forthcoming. The persistence of a murmur after a febrile attack, or its appearance, should be considered along with the other evidences. Thus, after the subsidence of the fever, if the rate becomes slow and the youthful type of irregularity is developed, it may be assumed that the heart has escaped infection, and that the murmur is functional. If, on the other hand, the rate is persistently increased, then the fear is that the heart has become infected, and that active changes are going on. The importance of this aspect of the subject is more fully dealt with in Chapter LXI.

The significance of so-called functional murmurs.—Seeing that individuals with these murmurs may be in perfect health, leading strenuous lives, and never showing any sign of heart failure, we may conclude that murmurs may be a physiological and normal sign, and indicate no impairment of the heart's efficiency, nor foreshadow the oncoming of heart failure.

I wish to insist upon this fact, for the possibility that a healthy heart may present a murmur is so opposed to the views of a great many of the profession, teachers and practitioners that much harm is done to people who have to be examined for life insurance or for entrance to the services. I base my views on the fact that I have watched numbers of healthy young people, who exhibited such murmurs, grow up into manhood and womanhood, and lead healthy and vigorous lives, and never show the slightest sign of heart failure. Moreover, if we consider how little we know of the causation of murmurs, it will be realized how little justification we have for treating them as abnormalities. Even if they were due to regurgitation, it is quite conceivable that the regurgitation might be an essential to a good functioning heart. We might reason as follows. A chamber of the heart will contract most efficiently when it has reached a certain degree of distension. If circumstances should arise, as during effort, when a chamber was overdistended, then the conditions would not be favourable to a good contraction. But if a mechanism were present, which relieved the chamber which was distended, a slight regurgitation might take place, a murmur might arise and the contraction be effective. I have already pointed out that the fear of regurgitation has attained the dimensions of a bogy in the medical mind ; further, I have already pointed out that, even with organic disease, the amount of regurgitation required to produce a murmur is very slight. Functional murmurs may be present in debilitated individuals, and in these cases the murmur is not necessarily an indication of disease or impairment, nor is the valve incompetency which its presence indicates the cause of the debility. Manifestly, the murmur is but one of the many symptoms which the individual may exhibit, and the diagnosis of such cases will necessitate a search for other evidences which may throw light on the cause of the debility. A functional murmur may be present in cases of grave heart failure, but in such cases the murmur is but an associated symptom ; for the valvular incompetence is not the cause of the heart failure, but is

merely one of the changes which have arisen in consequence of impaired functional efficiency of the heart muscle.

The significance of organic murmurs.—Organic murmurs have a varied significance, and it is necessary to grasp the idea that, though murmurs are associated with heart failure, the valve lesions which they indicate are not the cause of it. It is also well to bear in mind that individuals with murmurs, due to damaged valves, caused by some infective disease, such as rheumatic fever, may lead vigorous lives and be engaged in strenuous occupations, and never show any sign of heart failure. From this we can conclude that valve lesions in themselves may not necessarily be of serious significance. As, however, murmurs are often associated with heart failure, the presence of organic murmurs should be considered from the following standpoints: (1) as an indication that the heart has been invaded by a disease process; (2) whether the damage done by the disease process is stationary or slowly progressive; (3) whether the disease process has affected and impaired the heart muscle at the same time; and (4) whether the damage to the valve is such that it obstructs the work of the heart, so as to embarrass the muscle sufficiently to impair its efficiency. In acute illnesses, it will not be possible to answer these questions, for there the immediate question concerns the acute affections. In chronic cases, that is to say what is called chronic valvular disease, it is absolutely necessary that these questions should be considered and answered if a rational conclusion is to be obtained. The manner of obtaining the knowledge necessary to answer these questions will be dealt with in the description of the different valve lesions.

The estimation of the significance of murmurs is not based on the murmur itself, but on the functional efficiency of the heart, and on the presence or absence of other signs of cardiac affections (size, rate, and rhythm). If we find in a heart of normal size and rhythm (or with the 'youthful type' of irregularity) a systolic murmur with absence of any sign that would indicate that it is definitely organic in origin, and with a good functioning organ, then we may conclude that the heart is perfectly normal. If there be evidence of weakness, or other signs of abnormal conditions present, then the opinion should be based on these other signs, and not on the murmur.

CHAPTER LVI

Valvular Defects

The manner of heart failure with valvular defects.
Mitral stenosis.
Conditions inducing heart failure in mitral stenosis.
The murmurs present in mitral stenosis.
The mid-diastolic murmur.
Progress and symptoms of mitral stenosis.
Occasional symptoms.
Mitral regurgitation.
Murmurs due to mitral regurgitation.
Conditions inducing heart failure in mitral regurgitation.

The manner of heart failure with valvular defects.—It is manifest that valvular defects can embarrass the work of the heart in two ways: first, by narrowing the orifice and thereby impeding the outflow; second, by imperfect closure so that leakage occurs.

The defects are recognized clinically mainly by the presence of murmurs; but it must not be assumed that absence of murmurs implies an intact valvular apparatus, for great widening of an orifice and large regurgitation may take place when no murmur can be detected. As valvular lesions are produced by a variety of conditions, it might have been more logical to discuss them under the heading of these conditions. As, however, they are presented to us at a stage when the immediate symptoms of their causal condition are in abeyance, it is more convenient to describe them at the time that the heart-changes evoke symptoms of heart failure. Years may elapse after the mischief has been done to the valves, before symptoms arise that call attention to the heart trouble. In the acute condition producing the valvular lesion, the matter is presented in an entirely different aspect, for the febrile state and the symptoms associated with the cause of the fever predominate. In chronic valvular affections the subjective symptoms of heart failure only arise when exhaustion of the heart muscle sets in. The symptoms due to exhaustion appear at varying periods, after the damage has been done to the valves; and the time of the appearance of these symptoms depends on the degree of embarrassment offered to the heart's work by the damaged valve, on the condition of the muscle-wall, and on such accessory factors as tend to exhaustion, as over-exertion, excessive food, drink, and so forth.

In organic lesions of the valves it must always be borne in mind that the

sclerotic process causing the lesions may be progressive, and that there may also be present advancing changes in the heart muscle.

Generally speaking, the symptoms of heart failure show little that is distinctive of the particular valves affected. In the aortic cases, the reflex sensory phenomena are more prominent, and the ashen colour of the face is sometimes characteristic. Where there is a mitral lesion, the pulmonary symptoms are usually more prominent, and the face may be ruddy with a dark tinge. Apart from such differences, there is a great similarity in the symptoms of heart failure produced by all kinds of lesions.

Mitral Stenosis

Conditions inducing heart failure in mitral stenosis.—This is, perhaps, the most common of valvular defects with which heart failure is associated. It arises generally in consequence of rheumatic endocarditis, though it may be found in people with no rheumatic history, and a previous history of erysipelas, or some other febrile complaint, may give a possible clue to its origin.

The condition is never recognized during the acute process which induces it, for the reason that the stenosis is not produced till the cicatrizing process following the inflammation narrows the orifice, and on account of its origin in scar-formation it is often a progressive lesion. Once the stenosis is present it may remain moderate in amount, and offer so little embarrassment to the heart that patients may reach extreme old age with no heart failure. As a rule, however, the cicatrizing process goes on with varying rapidity until in some cases the mitral orifice is reduced to a mere slit, and the valves resemble a thickened calcareous diaphragm. It is important to bear in mind the progressive nature of the lesion, for it accounts for the varying changes in the symptoms. It should also be borne in mind that the cicatrizing process may be going on in the muscle, causing contraction of the chordæ tendineæ, impairing at other places the functional activity of the heart muscle, and affecting the genetic system, may interfere with its function, produce heart-block, auricular fibrillation, and other abnormal actions, thereby profoundly affecting the efficiency of the heart and modifying the nature of its rhythm.

From this, it can readily be understood that the manner in which heart failure is brought about in many cases is somewhat complicated. In some, embarrassment may not ensue until the narrowing of the orifice has become extreme. In others, there may be a fatal issue while the narrowing is yet moderate. In the latter cases, the muscle-wall will inevitably be found to have been damaged.

The murmurs present in mitral stenosis.—*The presystolic murmur.* The presystolic murmur—the auricular systolic murmur of Gairdner—is due to the contraction of the left auricle forcing blood through the narrowed mitral orifice. With the varying changes in advancing cicatrization, the murmurs of mitral stenosis alter, and present peculiarities that have hitherto

not been sufficiently appreciated. In the very early stages—some years before the appearance of a murmur—I have detected a slight presystolic thrill. The first murmur to appear precedes, or runs up to, and seems to terminate in the first sound, and is audible over a small area around the apex. At first it may not be always present. This murmur may vary in duration, being usually short and abrupt, but it sometimes begins earlier and is somewhat prolonged. It is of a crescendo character, rising in pitch till it ends in the first sound.

The diastolic murmur.—With advancing stenosis of the orifice, another murmur makes its appearance, namely, one occurring immediately after the second sound, heard only in the immediate neighbourhood of the apex-beat. At first it is very faint, and not very constant, but it usually increases in duration until the whole diastolic period may be filled up by it. This diastolic mitral murmur diminishes in intensity from the beginning — differing thus in its diminuendo character from that of the presystolic. Frequently we can detect a continuous murmur during the diastole of the heart, beginning loudly, falling away, then increasing in intensity. The first or diminuendo portion of such a murmur is the diastolic mitral murmur, while the terminal crescendo portion is the presystolic. The cause of the diminuendo diastolic mitral murmur is the flow of the blood that has been accumulated in the auricle, during the ventricular systole, through the narrowed mitral orifice ; this begins as soon as the mitral valves open, that is, when the pressure in the ventricle falls below that in the auricle. These changes in the murmurs are diagrammatically shown in Figs. 260 and 261.

The disappearance of the presystolic murmur.—The next change in the character of these murmurs is the sudden disappearance of the presystolic crescendo murmur, while the diastolic murmur persists. Usually this change is followed by the onset of grave symptoms of heart failure, the heart's action becoming rapid and irregular. At other times the change takes place with no serious symptom, but the heart invariably becomes irregular. I have explained this fully in Chapter XXVIII., as being due to the fact that the rhythm of the heart starts no longer with an effective auricular systole, but the auricle is standing still in the condition we call fibrillation.

When the heart's action is slow, there is no difficulty in recognizing the diastolic murmur, and the absence of the presystolic. The diastolic murmur is sometimes of great length, starting immediately after the second sound, and when the heart's action is rapid, it may fill up the whole diastolic pause, and it might hastily be assumed that the murmur was presystolic. But if it be carefully auscultated, it will be found that it is not crescendo in character ; when a longer pause occurs, it will be found that the murmur stops short before the first sound, so that there is a silence between the end of the murmur and the first sound (see Figs. 260 and 261). In these cases the jugular and liver pulses are invariably of the ventricular form.

The mid-diastolic murmur.—Although the usual position in the cardiac cycle of the presystolic murmur is immediately before the first sound, there

are a few cases in which it does not terminate abruptly in the first sound, but is separated from it by a very brief interval (see Observations 7 and 67). In some of my cases I asked a number of my colleagues to mark out on a tracing

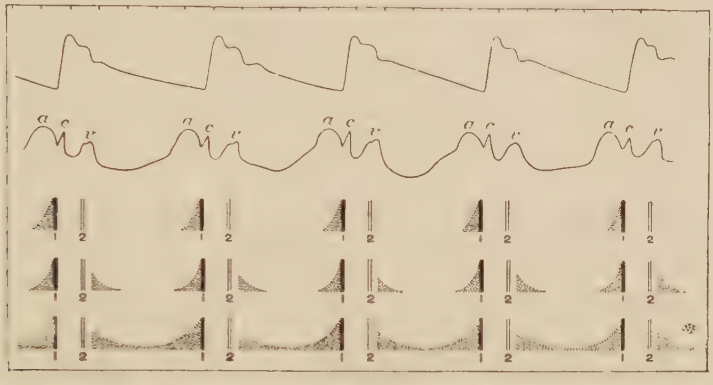


Fig. 260. Tracings of the radial and jugular pulse with diagrammatic representation of the changes in the murmurs of mitral stenosis. The downstrokes 1 and 2 represent the first and second sounds of the heart. The first indication of stenosis is the appearance of the murmur before the first sound, and on reference to the jugular tracing this murmur will be found to coincide in time with the wave *a*, due to the systole of the auricle. With an increase in the stenosis, a short diastolic murmur appears, immediately after the second sound, and this is represented in the middle diagram. A further increase in the stenosis, shown in the lowest diagram, leads to the prolongation of the diastolic murmur, which in combination with the presystolic murmur fills up the whole interval between the second and first sounds. (For the next change see Fig. 261.)

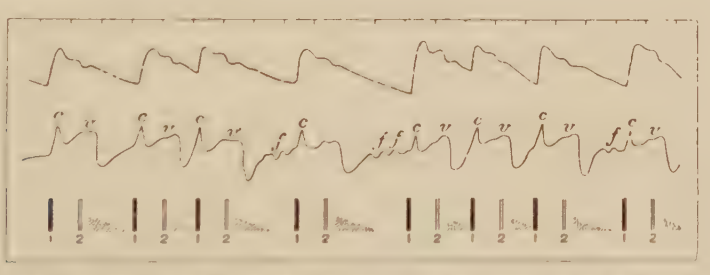


Fig. 261. After the changes described in Fig. 260, the next change is a sudden alteration of the rhythm of the heart. In Fig. 260 the heart is quite regular. In Fig. 261 it is irregular. The wave *a*, which is a marked feature in the jugular tracing of Fig. 260, is absent in Fig. 261. The presystolic portion of the murmur is also absent in Fig. 261, this feature being best appreciated during the longer pauses of the irregularity. The disappearance of these evidences of auricular activity and the irregular action of the heart indicate the onset of auricular fibrillation. The waves *f f* are due to the fibrillating auricle.

of the radial pulse the exact position of the murmur in the cardiac cycle, and every one without hesitation indicated the position where the loudest part of the murmur is separated by a distinct interval from the pulse-beat. When a jugular or an apex tracing was taken, it was seen that the position

was identical with that of the auricular systole. In other words, there was a delay in the transmission of the stimulus for contraction between auricle and ventricle. This delay can sometimes be increased by digitalis, and the position of this murmur in its relation to the first sound moves in the same way; such a murmur has hitherto been described as '*mid-diastolic*'. A mid-diastolic murmur therefore indicates not only the presence of mitral stenosis, but also delayed conduction between auricle and ventricle.

Progress and symptoms of mitral stenosis.—From the progressive nature of the lesions in the valve and in the heart muscle, it will be realized that the symptoms are not constant.

The patient seeks the doctor's help mostly in early or middle adult life. The complaints then are shortness of breath, a sense of suffocation, and palpitation on exertion. In some the face is ruddy, with a hue a shade darker than is compatible with the ruddy countenance of robust health;

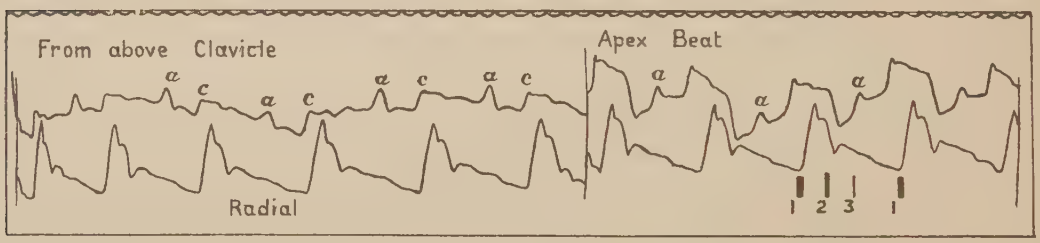


Fig. 262. Simultaneous tracings from the movements in the neck and the radial in the first part of the tracing, and of the apex and radial in the second part of the tracing. The auricular waves (*a*) from the neck and from the apex are seen to be widely separated from the ventricular contraction. The first and second sounds are represented with a muffled sound at the time of the auricular wave.

in others there is pallor. At this stage there is little or no increase in the size of the heart and no dropsy. A presystolic murmur can usually be detected. The patient's complaints may be the only evidence we have of the heart failure, and these point to an exhaustion of the reserve power of the heart. After a period of rest, this exhaustion may disappear, and the patient may go on for years with but little further trouble. After a time, however, some again break down, and the symptoms complained of may be of the same nature. Frequently, however, a change is found in the character of the murmurs—a diastolic murmur usually being perceived, and there is sometimes a longer duration of the thrill, these signs implying an increased narrowing of the orifice. On the other hand, in those in whom no further narrowing takes place, the murmur does not change, and the patient may go on for many years, and, if a female, may bear children, with no breakdown. In these cases we can infer that there is no progressive muscular or valvular sclerosis. With the increased narrowing of the orifice, as indicated by the appearance of the diastolic mitral murmur, the heart becomes more em-

barrassed, the symptoms become much more distressing, and finally dilatation of the heart may set in. But even without the progressive narrowing, dilatation may appear early, and then it may be inferred with certainty that the rheumatic process has permanently injured the heart muscle.

The rhythm of the heart may become continuously irregular, and with the onset of auricular fibrillation further embarrassment arises, as described in Chapter XXXIII. If there be no change in the size of the heart with auricular fibrillation, and no great acceleration of rate, the heart failure may be very slight in degree ; but if the heart dilates, especially if the rate is accelerated, then all the extreme symptoms of heart failure follow (dropsy, enlargement of the liver, etc.).

In the vast majority of cases the heart recovers from its first breakdown, and usually from many subsequent attacks. Indeed, after one attack, I have known patients go on for twenty years and more, with no further trouble beyond a slight limitation of the field of response to effort.

After repeated attacks, the patient's life becomes one of great limitation. The future depends often on the rapidity of the advance of the sclerotic process of the valves and the heart muscle. If the rate of advance be slow, and the heart muscle capable of responding to treatment, the patient may go on for many years with a crippled existence. Sometimes we find in the young, about 20 years of age, at the post-mortem examination, the orifice narrowed to a mere slit. In others we find the mitral orifice not much contracted, but the heart-wall greatly dilated, and evidence of fibrosis of the muscle. Hence it will be seen that the progress of these cases is largely dependent on the rate of change in the muscle as well as in the valve. The final issue is usually by great extension of the dropsy and exhaustion.

There are several complications which may arise.

Occasional symptoms.—*Paroxysmal tachycardia.*—In place of the auricular fibrillation being permanently established, it may appear intermittently, as attacks of paroxysmal tachycardia. Other conditions may produce these attacks of paroxysmal tachycardia, notably auricular flutter. These attacks are of varying importance. Some patients may have them for more than twenty years and seem little the worse. Others may have them occasionally, and then the heart settles down with the rhythm permanently altered. In such cases the future depends on whether the heart slows down or remains at a greatly increased rate, as already described.

Hæmoptysis.—At various stages, patients may be seized with great bleeding from the lungs. Here doubtless the cause is engorgement of the pulmonary circulation and rupture of the blood-vessels. As a rule, this is a grave sign, the patient dying sometimes shortly after an attack, although in auricular fibrillation the patient may make a good recovery, especially if the heart is susceptible to digitalis.

Cerebral embolism.—Vegetations may exist at the mitral valves, with no certain sign of their presence until a small portion is detached and impacted in some vessel, giving rise to a hemiplegic attack, or an attack of

aphasia. Usually, recovery takes place speedily, and may be permanent, but cases have been recorded in which the aphasia or hemiplegia has remained complete for many years. Clotting may occur in the auricular appendage during auricular fibrillation, and be a source of embolic infarct.

Attacks of angina pectoris.—Although very rare, these may occur in mitral stenosis. In the few cases I have observed, they were all secondary to some excessive exertion, and the patients had only one or two attacks, remaining perfectly free from them for years afterwards.

Mitral Regurgitation

Mitral regurgitation may be the result of a damaged valve, or of dilatation of the orifice, from weakness of the muscles supporting the valves.

Murmurs due to mitral regurgitation. — The murmur of mitral regurgitation is systolic in time, heard loudest at the apex. It may be soft and blowing, of little intensity, and heard over a very limited area, or propagated into the axilla ; or it may be rough and loud, and heard over the whole heart and round to the back of the chest. It is not always possible to tell whether it is due to dilatation of the orifice or to damage of the valves. The rough loud murmur with an accompanying thrill is always a sign of damaged valves.

Conditions inducing heart failure in mitral regurgitation.—When the muscle is unimpaired, little or no bad effect may follow damage to the mitral valves. Even where the regurgitation is due to ' functional ' dilatation of the orifice from depressed tonicity, the contractile power of the muscle may maintain a good and efficient circulation. The really serious trouble in connexion with mitral regurgitation arises when the muscle is impaired and the regurgitation is due to a complication of the dilated orifice and diseased valve. The subsequent results depend on the degree of the exhaustion of the muscle of the heart. The backward pressure resulting from the regurgitation embarrasses the left auricle. The exhaustion also affects the right ventricle and so adds to the embarrassment of the pulmonary circulation. While back-pressure is a factor of importance and may be a predisposing cause, yet it produces comparatively few symptoms until dilatation of the heart occurs. The dilatation is generally looked upon as the result of the regurgitation, the back-pressure ultimately producing yielding of the walls of the right heart. This is not quite correct, for long before there is any back-pressure we may find evidence of a dilated right heart. If we examine carefully the condition of the heart when the valves have been damaged by rheumatic endocarditis, during one of the slight attacks of heart failure which are liable to occur after over-exertion, we may find the heart slightly dilated, the right ventricle being in front so that the left ventricle is pushed to the left behind the lung ; the apex beat is then due to the right ventricle, which gives a negative cardiogram (Fig. 36). After a few days' rest and treatment the right heart may retreat and the apex beat is then due to the left ventricle ;

the cardiogram now presents the normal characters, rising during systole. In such cases there is no evidence whatever of pulmonary engorgement and back-pressure. In fact, in the majority of cases, as Graham Steell says, ' the change in the valves is altogether inadequate to explain the evidently free regurgitation that occurred during life, and the disastrous dilatation of the heart. The muscle-failure factor, it may be presumed, was the essential one.'

The damage to the valves is most commonly the result of rheumatic endocarditis, and, as we have seen, the process is rarely limited to the endocardium, but invades the myocardium. Septic endocarditis may also damage the valves. In all cases of mitral stenosis there is mitral regurgitation, but the amount of the regurgitation is never so marked as to be the serious factor in the case.

It will thus be seen that the symptoms produced by mitral incompetence are only of gravity when there is also muscle failure, and this is dealt with in sufficient detail in Chapter XLVII.

CHAPTER LVII

Valvular Defects (*continued*)

Affections of the tricuspid valves.
  Tricuspid incompetence.
  Tricuspid stenosis.
Disease of the aortic valves.
  Ætiology.
  Aortic stenosis.
  Aortic regurgitation.
Prognosis in valvular affections.
Treatment.

Affections of the Tricuspid Valves

Lesions of the tricuspid valves are rare, and are nearly always associated with similar lesions in the mitral and aortic valves. The heart failure associated with these lesions is never due to the tricuspid lesion alone.

Tricuspid incompetence.—Although actual disease of the valves is rare, incompetence of the tricuspid orifice is extremely common—so common, indeed, that I am inclined to look upon the valves as being barely able to close the orifice perfectly. This view is based upon the observation of many patients, in whom I have been able to detect a tricuspid systolic murmur with no appreciable increase in the size of the heart. The murmur in many cases is very fugitive, being present in the first few minutes of an examination, and disappearing when the heart becomes quieter. A consideration of the size of the orifice and the size of the valves led John Hunter to doubt their competency, while Mayo declared that the tricuspid valves never perfectly close the orifice. Experimentally, it has been found impossible to raise the pressure in the right ventricle, on account of the ease with which regurgitation takes place through the tricuspid orifice.

The slighter forms of tricuspid murmurs are limited to a small area over the middle of the sternum. With increase in the size of the right heart, they may be heard over the whole anterior surface of the heart. They are often associated with mitral systolic murmurs, but one can usually detect a difference in quality in the mitral murmur heard beyond the left nipple line and in the axilla, from the tricuspid murmur heard over the middle of the sternum.

It should never be concluded that no tricuspid regurgitation occurs

because of the absence of a murmur, for it is of frequent occurrence to find evidence of tricuspid incompetence in the character of the jugular and liver pulsation (ventricular form), and in the greatly widened orifice post mortem, while during life there was no systolic tricuspid murmur. A weak muscular wall and a wide orifice may give rise to no murmur.

I dwell at some length on these points, not because the tricuspid regurgitation is of much practical value, but because misunderstanding of its presence has led to a wrong construction being put on the effects of tricuspid regurgitation, and to the real significance of the ventricular form of the venous and liver pulse being missed. I have already pointed out that slight regurgitation in a normal heart would add to the accumulating blood in the right auricle during ventricular systole, and would, therefore, be a factor in the production of wave *v* in the jugular pulse. Now most writers overlook the fact that a dilating auricle is interposed between ventricle and jugular,

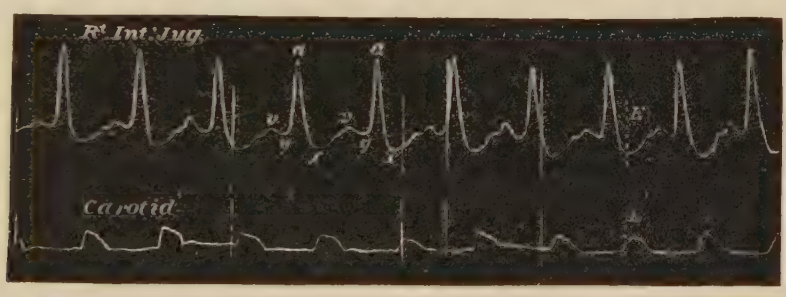


Fig. 263. Simultaneous tracings of jugular and carotid pulses showing a large wave (*a*) due to the auricle. Compare with Fig. 94. (Observation 4.)

and have assumed that as soon as tricuspid regurgitation takes place a wave appears in the jugular at the beginning of ventricular systole. Fig. 263 shows that this is not so, for in this tracing the *v* wave appears at the end of the ventricular systole. The ventricular form of the venous pulse has been regarded as only a sign of tricuspid regurgitation, and the real significance of this very important symptom has been missed. That it is a sign of tricuspid regurgitation there is no doubt, but it is a sign of far greater significance, namely, that the auricle does not precede the ventricle in the cardiac cycle.

Tricuspid stenosis.—In the majority of cases, tricuspid stenosis is not recognized during life, as the symptoms produced are not always distinctive. It is only rarely that a presystolic tricuspid murmur is heard; I have only heard it in three cases, in which it was present in a very limited area over the middle of the sternum. There is usually present also a mitral presystolic murmur at the apex; but as each murmur is confined to such limited regions I have had no difficulty in distinguishing them. In one case, the auricle had become so greatly hypertrophied that it sent back a large wave into the jugular, and that with such force that it caused the valves in the jugular

and subclavian veins to close with a snap, which I could hear over these veins as a clear, sharp sound preceding the first sound.

As a result of the stenosis of the tricuspid orifice, the right auricle hypertrophies, and on this account sends a wave back into the vein with such force that it distends the liver, and I therefore look upon pulsation of the liver with a marked wave due to the auricle, as a sign suggestive of tricuspid stenosis.

Disease of the Aortic Valves

Ætiology.—By far the greater number of cases of affection of the aortic valves owe the lesions, primarily, to rheumatic or syphilitic endocarditis and the sclerotic process accompanying arterial degeneration. Under rare circumstances the valves may rupture, but here there is usually some antecedent disease of the valve. Congenital defects may, in rare cases, give rise to great embarrassment of the heart.

It is the lesions induced by the first-named conditions that require most consideration. The condition is usually well established before it is found out. In many cases, the presence of aortic changes is discovered accidentally, when a systematic examination is being made for other ailments, or for life insurance or a health certificate.

The heart failure in aortic valvular disease is rarely due to this lesion alone. In the majority of cases, changes impairing the power of the muscle have been proceeding at the same time as those that induced the valvular changes.

In the rheumatic cases, there is frequently present a complicating lesion of the mitral valve.

When the valvular disease embarrasses the heart's work by the extent of the lesion, as by great incompetence, and the heart muscle is healthy, the latter responds to the obstruction to its work by hypertrophy, and this may proceed to an enormous extent, giving rise to one of the largest of human hearts—the cor bovinum.

Aortic stenosis.—Aortic stenosis is often associated with aortic regurgitation, and the symptoms of the latter usually dominate the situation. When there is little or no regurgitation, the symptoms of aortic stenosis, being less prominent, are often only detected accidentally in the routine examination of the patient.

The sign most characteristic of aortic stenosis is a murmur systolic in time, heard loudest over the second right costal cartilage, and propagated into the carotids. It may be faint—a mere whiff, or it may be prolonged, and accompanied by a thrill perceptible over the upper part of the chest-wall. The heart's rate is often slow, between fifty and sixty beats per minute. The radial pulse is sometimes very characteristic. It impinges against the finger in a slow, leisurely fashion, and a sphygmographic tracing may show a slanting upstroke with a slight interruption near the summit (anacrotic pulse, Fig. 264), or even a double wave at the top (pulsus bisferiens,

Fig. 265). Graham Steell and Lewis state that they have been able to detect this double beat by the finger, and Graham Steell says he has perceived it on one side only. Its real nature is yet obscure.

Beyond these signs, there is little that is characteristic in aortic stenosis. There may be symptoms of angina pectoris, but these are due to associated changes in the heart muscle, and other evidences of heart failure can be referred to the same cause.

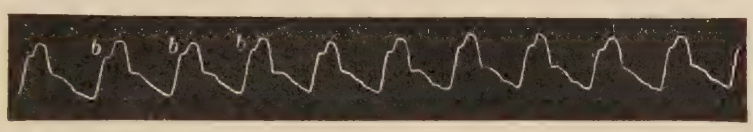


Fig. 264. Anacrotic pulse, from a case of aortic stenosis.

It is necessary to emphasize the fact that a systolic murmur at the base is frequent in healthy hearts. Unfortunately it is too often looked upon as a sign of aortic disease. When there is only this murmur present—without thrill, and the pulse is not anacrotic—it may be safely assumed that the murmur is not aortic in origin, but physiological, or so slightly pathological as to be of little significance. This is especially the case in the young. After middle age a systolic murmur in this region is not uncommon and is of little or no significance.

Aortic regurgitation.—The aortic valves being contracted are no longer able to support efficiently the column of arterial blood during diastole, but permit a backward flow into the heart, and as a result we find certain alterations in the character of the second sound and of the arterial pulse.

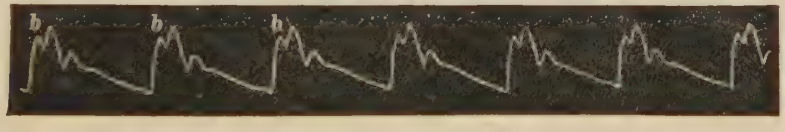


Fig. 265. Pulsus bisferiens, from a case of aortic stenosis.

The closure of the valves no longer gives to the second sound the characteristic snap, but the sound ends in a murmur sometimes long drawn out, sometimes so brief as to be scarcely perceptible—as if the second sound terminated not abruptly but with a faint sigh. The diastolic murmur is usually propagated down the sternum, but sometimes it is heard loudest at the apex. Foster has suggested that this variation in the propagation of the murmur depends on the direction given to the backward flow by the position of the retracted cusp. This seems plausible, but I have not been able to verify it, and the explanation is ignored in recent text-books.

The regurgitant murmur is usually associated with the murmur of aortic stenosis, and we get the characteristic double aortic murmur (bellows

murmur). There is frequently dilatation of the smaller arteries, and this, combined with the effect of the regurgitation on the arterial pulse, causes the artery to become emptier than usual towards the end of diastole. This means a fall of pressure, and in order to maintain a normal mean pressure, the heart increases the force of its contractions, raising the pressure during systole, so that there is a great increase in the systolic pressure and a great fall during diastole, thus giving rise to the characteristic collapsing pulse (Corrigan's pulse, the water-hammer pulse, Figs. 266, 267, 268). The collapsing character of the radial pulse may be intensified by raising the arm above the head.

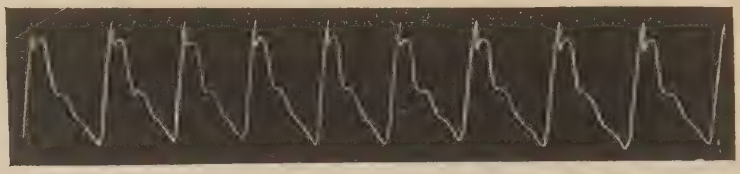


Fig. 266. Pulse of slight aortic regurgitation with good heart muscle.

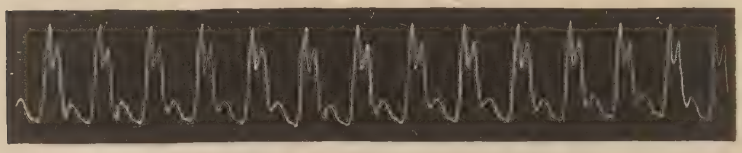


Fig. 267. Pulse of slight aortic regurgitation with great cardiac failure.

Fig. 268. Pulse of extreme aortic regurgitation with great cardiac failure.

At times, the arterial pulse is conveyed through the capillaries into the veins, and G. Gibson has obtained a graphic record of such pulsation of the veins on the back of the hand. If the forehead be rubbed so as to produce redness, the flush is seen to wax and wane with each beat of the heart (capillary pulsation).

The double aortic murmur may be detected without any history of a heart affection. In many cases there may be little or no dilatation, and the individual may be able to indulge in games and in occupations requiring considerable exertion with no discomfort. In such cases it may safely be assumed that the damage to the valves has been slight, and that the heart muscle has escaped serious injury.

In other cases the heart is greatly enlarged, and the apex beat is diffuse

and forcible. The systole and diastole of the heart may cause movements of the liver that simulate pulsation of that organ, but analysis of its graphic records shows it to be merely the dragging up and pushing down of the liver by the changes in the size of the heart (Figs. 299, 300). Even under these circumstances the individual may for years pursue an active vocation, but he is always liable to attacks of heart failure. The condition is often associated with affection of the mitral valves ; and this is one of the factors participating in the production of the heart failure that finally terminates these cases.

The most frequent sufferers from aortic valvular disease are the middle-aged. In them the sclerotic process has been gradually advancing, and the early symptoms of exhausted reserve force have been neglected until symptoms of distress command attention. There may have been a history of rheumatism, of excessive drinking, of hard bodily exertion, of syphilis, but, on the other hand, no definite causal condition may be discovered. The facial aspect is frequently pale grey (earthy countenance), though in others it may be full-blooded and ruddy. The complaints are varied. Shortness of breath on exertion, violent throbbing in the neck, attacks of pain over the chest on exertion, are amongst the most common symptoms of which the patient complains in the first instance. For a varying period, under suitable treatment, a certain store of reserve force is gained, and he may go on for months or years, sometimes in fair comfort, but his existence is usually more or less crippled.

A feature strikingly characteristic of aortic regurgitation is the susceptibility to nerve stimulation. The heart and blood-vessels are readily stimulated, the former to rapid action, the latter to variation in the calibre of the vessels. The recognition of this peculiarity is of importance ; for I have repeatedly seen individuals in whom the heart became rapid in action, and with consequent exhaustion, when they were first informed they had got an affection of the heart. Moreover, these cases are very susceptible to a mental stimulus, which may increase the heart-rate, and also raise the blood-pressure and induce attacks of angina pectoris. The difference of the blood-pressure in the arteries of the arm and leg is commented on (p. 455), and also the susceptibility to attacks of pain (p. 335).

The end of these patients is very varied, and I am far from comprehending the mechanism of heart failure in many. Many aortic cases suffer from extreme cardiac exhaustion, and show no objective signs of heart failure, such as dropsy. Where there is dilatation and dropsy, it is generally because there is present some disease of the muscle wall. Some of the most intractable cases of auricular fibrillation are those where there is also aortic regurgitation, so that I look upon the appearance of auricular fibrillation in aortic cases as a somewhat grave event, more so than when it occurs with mitral stenosis. I have seen a few cases die during a sudden attack of dyspnœa of the greatest severity.

Prognosis in valvular affections.—Let it always be remembered that

frequently sound and healthy hearts may show a murmur, and that it is necessary, therefore, to seek for other evidences on which to base a prognosis. The heart failure which may be present depends upon so many and so varied conditions—as the extent of the valvular lesion, its progressive nature depending on the cicatrizing process affecting the valves, the coincident changes in the muscle and in the genetic system, the conditions of life of the individual—that no rule applicable to all cases can be made. If, however, an attempt be made to appreciate the value of the symptoms present, on the lines I have laid down, an approach to a true prognosis may be made in each case. There is just one point I wish again to insist upon : let no single symptom be the ground for forming an unfavourable prognosis. In this respect, the presence of a murmur has so obsessed the profession that a vast amount of positive harm is continually being done to patients, by taking too seriously the prognostic significance of this sign. The field of cardiac response is the only true and safe guide in these cases. Even if for the time being it is limited, judgment should be suspended until an opportunity has been obtained for ascertaining to what extent the heart muscle can regain a store of reserve force.

Treatment.—As heart failure with valvular defects touches every phase of the subject, the matter of treatment must be discussed from a very wide aspect. The special chapters on treatment, therefore, include the full consideration of this subject.

CHAPTER LVIII

Adhesive Mediastino-Pericarditis

Ætiology.
Symptoms.
Prognosis.
Treatment.

Ætiology. — The adherent pericardium, secondary to rheumatic pericarditis, rarely gives rise to any sign. In these cases the pericardium is not adherent to structures outside the heart. On the other hand, certain obscure inflammatory affections, probably of a tubercular nature, such as occur in ' polyserositis ', give rise to very marked phenomena. There is an extension of an inflammatory process which affects all the structures in the mediastinum, welding together the heart and pericardium, and firmly binding these to all the surrounding structures. The heart becomes anchored to the spinal column behind and to the chest-wall in front. As the spinal column is unyielding, the contracting heart pulls on the ribs in front, and as they yield to a greater or less extent we find the ribs drawn in during systole and springing back during diastole. This embarrassment of the heart leads to great enlargement in its size, and some of the biggest hearts met with are due to this disease.

Symptoms.—The patients are always very short of breath, and as the disease advances they have to be propped up when in bed. As a rule, little or no pain is complained of, but in one case I found that attacks of angina pectoris of the most severe form were easily provoked. A slight effort would bring on an attack, especially if the patient laughed. Attacks could sometimes be induced by pinching the skin under the left nipple, or by applying the stethoscope.

The adhesion of the heart to the lungs, blood-vessels, and other surrounding structures produces a great variety of symptoms, the cause of many of which is obscure. The chief symptoms are great enlargement of the heart—so great sometimes as to cause a marked difference between the two sides of the chest—and retraction of the structures surrounding the heart during ventricular systole. The systolic retraction alone is not distinctive, as it occurs when the anterior surface of the heart is made up of the right ventricle (Fig. 246). During the ventricular systole there is often an indrawing of the lower intercostal space on the left side behind (Broadbent's sign, Fig. 269).

Tallant and Cooper have shown that this may arise in enlargement of the heart (with compression of the lungs) without pericardial adhesion. In such cases, however, the interspaces affected vary with respiration, and Cooper suggests that when they do not vary with respiration the sign is, as Broadbent states, an evidence of pericardial adhesion. When the chest-wall is thin and the heart is not covered by lung, the systolic retraction of the different interspaces can be seen in a peculiar wave-like rhythm.

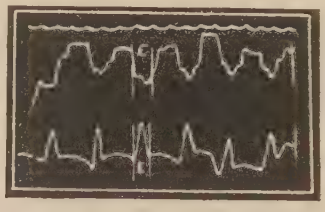


Fig. 269. The upper tracing was taken from the 9th left intercostal space behind from a case of adhesive mediastinitis, and shows ' Broadbent's sign ', which is seen to be an indrawing of the intercostal space during ventricular systole (space *E*). (Observation 43.)

Though various murmurs and modified sounds are often heard, no distinctive sign can be found on auscultation. The veins of the neck may sometimes be seen to swell up during inspiration. A very curious symptom is a sudden collapse of these veins at the beginning of diastole, which Friedreich has explained as due to the springing back of the ribs after the ventricular systole has dragged them down, the cavity of the thorax being thus suddenly enlarged and expediting the flow from the overfilled veins. This is well seen in the jugular tracing, Fig. 270, where the fall *z* is due to the diastolic expansion of the chest.

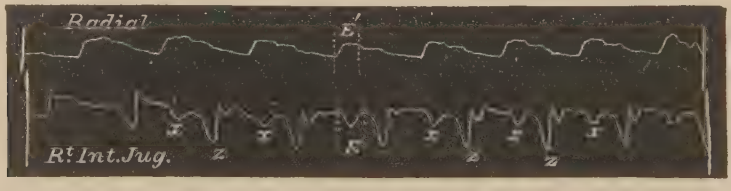


Fig. 270. Simultaneous tracings of the radial and jugular pulses, showing a great depression (*z*) occurring during the ventricular diastole. (From a case of adhesive mediastinitis.)

The radial pulse may show a diminution in size during inspiration—the pulsus paradoxus. It is now recognized that a respiratory variation of the pulse may occur in a great variety of conditions, but I think that in adhesive mediastinitis it presents certain distinctive features. Curiously enough, no tracings have been given, so far as I know, showing the pulsus paradoxus in adhesive mediastinitis along with the respiratory curve, except in two instances taken by Nicholson from Gibson's cases. The study of these

tracings, compared with some I have taken, leads me to think that the variations point to very different causes. As, however, my observations are few in number, I do not enter into the subject, but call attention to a field that needs exploring.

There is usually associated with these signs enlargement, and sometimes pulsation of the liver. The spleen also may be greatly enlarged, and there may be considerable dropsy.

Prognosis.—The future of these cases is bad, though they sometimes show periods during which they make remarkable progress towards recovery from serious symptoms. But these are only temporary, and they gradually drift to a fatal issue.

Treatment. — The treatment of these cases has so far been unsatisfactory, and one can only advise the principles usual in extreme heart failure. Attempts have been made, following the suggestion of Brauer, to free the heart by resecting the ribs. This has been done in several cases, and Wenckebach describes marked improvement in a case in which the operation was performed.

CHAPTER LIX

Congenital Affections of the Heart

Ætiology.
Symptoms.
Prognosis.
Treatment.

Ætiology. — Congenital heart affections are due to the persistence of certain fœtal forms of the circulation, such as persistent patency of the foramen ovale or ductus arteriosus, or to some interference with development leading to deformation of the valves, or narrowing and obliteration of the large arterial trunks. They may also arise in consequence of fœtal endocarditis. The conditions are incompatible with life in many cases.

It is only in exceptional instances that the symptoms permit of a recognition of the nature of the cardiac defect, and we are limited to the consideration of the manner in which the circulation is maintained, and the way in which the heart responds to effort, for our knowledge of most of these cases.

Symptoms.—When the circulation is embarrassed the most characteristic symptom is cyanosis, which is present in a great number of patients. Clubbing of the fingers is a common accompaniment of the cyanosis. The size of the heart is often greatly increased. This may be due to hypertrophy of the left ventricle, when there is an obstruction to the outflow of the blood through the aorta, or to dilatation of the right heart, when there is interference with the pulmonary circulation or a patent foramen ovale. Murmurs are usually present, almost invariably systolic in time, but it is often difficult to detect their origin.

There are two congenital defects of which I have had some experience. In one there is a loud systolic murmur heard all over the heart, usually loudest at the middle of the sternum. Usually the heart is not enlarged and the response to effort is good. I have no certain knowledge of the cause of this murmur but suspect a communication between the ventricles. I have watched several patients with the murmur grow up to their teens but have lost sight of them. One patient, a doctor 35 years of age, told me he had this murmur since childhood and he had suffered no embarrassment. Seeing how common (relatively) these murmurs are in childhood, and how rare in adult life, the question arises, Does the opening close and the murmur disappear in later life ?

In patent ductus arteriosus the communication between the aorta and pulmonary artery persists, and as the pressure is much higher in the aorta a continuous stream passes during the whole cardiac cycle from the aorta to the pulmonary artery, and, as Gibson has pointed out, this leads to a murmur which, beginning with great intensity at ventricular systole, extends over ventricular systole into diastole, fading away towards the end of diastole. This murmur is loudest over the second and third left interspaces, and here also a well-marked thrill synchronous with the murmur can be felt.

I have seen quite a number of patients—young girls—with this condition, but in only one over 30. This patient when first I saw her was pregnant, and my advice was sought in regard to the safety of labour and the use of chloroform. The heart's size was normal and the response to effort good, so I had no hesitation in allowing her to go to full term and have chloroform. This was done and she came through all right, and also through another pregnancy.

It is possible that here also the condition may disappear in later life, as I have never found it in people over 40 years of age.

Prognosis.—If there be no cyanosis, little or no enlargement of the heart, and the development of the child be good, with a fair field of cardiac response, then the prognosis is good ; otherwise the outlook is bad, though the child may lead a crippled existence for many years.

Treatment.—If the heart maintains the circulation well, no treatment is required. In more serious cases, beyond attending to the child's comfort and nourishment, special treatment for the heart is of little benefit, digitalis being rarely of value unless there is dropsy.

CHAPTER LX

Aneurysm and Dilatation of the Aorta

Aneurysm.
- Introduction.
- Definition.
- Cause.
- Arteries affected.
- The different kinds of aneurysms.
- Symptoms of aneurysm of the thoracic aorta.
- Aneurysm of the ascending aorta.
- Aneurysm of the transverse aorta.
- Aneurysm of the descending aorta.
- Aneurysm of the abdominal aorta.
- Prognosis.
- Treatment.

Dilatation.
- Symptoms of dilatation of the aorta.
- Prognosis.
- Treatment.

Aneurysm of the Aorta

Introduction.—In the chapter on capillaries it was suggested that the cause of degenerative changes was the obliteration of capillaries. The loss of the blood supply impaired the function of the organs as well as the nutrition of the structures. These degenerative changes affect the walls of the blood-vessels, and their functions become impaired and their structure damaged. These results are made manifest by the organs whose nutrition is interfered with, and this was shown to be the cause of angina pectoris. The changes which take place in the wall of the aorta are of the same nature as those which occur in other blood-vessels, but the results are different in that the walls give way under pressure and produce a variety of signs on account of their pressure on other structures.

Definition.—In certain diseased states of the arteries the walls give way and produce enlargements to which the terms aneurysm and dilatation are applied. By the latter term is meant a somewhat diffuse enlargement of the channel of a vessel for a considerable length; by the term aneurysm is meant a comparatively abrupt enlargement of a more circumscribed kind. It is not always possible, clinically, to adhere to this distinction between dilatation and aneurysm.

Cause.—Aneurysms and dilatation of the aorta are caused by the pressure of the blood within the artery on walls which have been weakened by the progress of degenerative changes, or by the effects of injury. The pressure causes bulging at points in which the resisting power of the vessels is impaired. Apart from injuries, aneurysms of the aorta arise from the degeneration of the arterial walls, in the majority of cases as a result of syphilis. It is far more common in males than in females, and in men it is more frequent among those who lead a life calling for strenuous muscular effort.

Arteries affected.—The aorta, especially the thoracic portion, is the artery which is most commonly affected; but all the larger arteries are liable, though the arteries smaller than the carotid are seldom affected except the smaller arteries of the brain, where miliary aneurysms are not uncommon in certain regions.

The different kinds of aneurysms.—Aneurysms of the aorta may be fusiform in shape or sacculated. In some cases the coats cannot be traced, in others the media is much atrophied, while the intima may be thickened and atheromatous. Laminated clot may partially or completely fill the cavity in the saccular variety.

A dissecting aneurysm is one in which the blood has forced its way between the intima and the media, or between the layers of the media, due to an injury of the intima, or as a result of its destruction owing to degenerative changes. It generally arises from the cracking across of an atheromatous patch. The blood, stripping the coats, may find its way back into the blood stream. Cases of so-called 'double aortæ' are dissecting aneurysms. As the result of a wound an artery may communicate directly with a vein, giving rise to the condition called aneurysmal varix. Where the communication is less direct, perhaps winding through the intervening tissues, the name of varicose aneurysm has been used.

Symptoms of aneurysm of the thoracic aorta.—It is rare that an aneurysm is discovered until it has advanced so far as to produce symptoms of pressure, and not infrequently its presence is unsuspected until it is revealed by an X-ray examination, taken, it may be, for other purposes, or at a post-mortem examination. One of the earliest signs in many cases is a pain, persistent and boring, referred to the back between the shoulder blades or over or below one shoulder blade. Occasionally pain is complained of in some other part of the chest or under the ribs. Even when the most careful physical examination is made no other symptom may be detected, but a persistent pain in the chest or its neighbourhood should always call, where possible, for an X-ray examination.

An X-ray examination is undoubtedly the surest means of recognizing an aneurysm, but while it is an easy means of diagnosis, yet there are cases where the most skilled radiologist may have a difficulty in deciding whether the shadow revealed is that of an aneurysm or a growth. The pulsation of the aneurysm is not always perceptible, and the aorta may communicate a movement to a tumour in contact with it. There is one point in which

caution is called for, and that is in determining whether there is a small aneurysm of the arch of the aorta. In some radiographs the shadow of the aorta shows a rounded swelling which is not infrequently mistaken for an aneurysm. This is really due to the angle at which the photograph is taken, so that the rounded swelling is the shadow of the arch and is a perfectly normal sign. It is called by some radiologists the ' aortic knob '.

Aneurysm of the ascending aorta.—When the aneurysm is small and close to the sinuses of Valsalva, there may be no sign. The aortic semi-lunar valves may be involved, and thus the cardiac signs obscure the aneurysmal. Anginal symptoms may be prominent when the coronary arteries are involved. Occurring higher in the arch, an aneurysm may attain large size. Pulsation may be visible or palpable to the right of the sternum in the second interspace. Slight pulsation detected in this area should lead to a more careful scrutiny for other signs. In aortic regurgitation or in excitement such a pulsation may be seen, but it also involves the carotids and other arteries. In aneurysm the pulsation is more localized and dullness will be detected on percussion over the pulsating area. The dullness may be central and extend a little to the right and left of the sternum. Sometimes a thrill or a double thrill may be felt, the second corresponding with the second sound of the heart. On auscultation a murmur may or may not be heard; occasionally a double murmur is heard. If the heart is hypertrophied and the semilunar valves competent the second sound may be accentuated.

When large the sac may erode the ribs and sternum. The superior vena cava is liable to be compressed causing the veins of the head and arms to be distended; the veinlets over the chest, particularly over the lower ribs on the right side, may become greatly dilated, and the flow of blood from the chest is then diverted and returns to the heart by the inferior vena cava. Rupture may take place into the pericardium, pleura, or superior vena cava.

Aneurysm of the transverse aorta.—The growth is generally backward and downward. It may, however, grow forward and become obvious as a pulsating tumour. An area of dullness may be detected in front or behind, near the spine. On pushing the finger deep into the episternal notch the pulsating aneurysm may be felt. Pressure symptoms predominate in aneurysm of this part of the aorta. Even though small the sac, growing backwards, may press on the trachea or a bronchus. The trachea in the neck may be pushed slightly but definitely to one or the other side. The trachea may be jerked down with expansion of the sac (tracheal tugging). One or other bronchus, generally the left, may be obstructed by pressure from the sac, leading to collapse and inflammatory changes in the lung, a not infrequent cause of death in this condition. Hæmorrhage from the sac weeping into the bronchus may precede a severe and fatal termination in this manner. Pressure on the œsophagus or on the vagi may cause difficulty in swallowing. Rupture of an aneurysm from the passage of an œsophageal bougie has caused death. Pressure on the left recurrent laryngeal nerve will give rise to

paroxysmal dyspnœa with stridor, and even attacks of loss of consciousness and death. Later a peculiar brassy cough develops with paralysis of the adductors of the glottis. The voice is altered, becoming hoarse, weak, or aphonic. Pressure on the sympathetic fibres leading to the neck will cause at first dilatation of the pupil of the eye on the same side, followed later by contraction when the impulses are blocked.

When the aneurysm occurs after the innominate has been given off but

Fig. 271. An X-ray photograph showing an aneurysm of the aorta after the innominate artery has been given off.

involves the origin of the left subclavian artery (Fig. 271), there may be a difference detected in the two radial arteries. This is usually described as due to a delay of the left radial pulse. As a matter of fact there is no delay but merely a diminished amplitude of the pulse-wave, and the wave slowly increases in height, whereas the increase in height in the normal right radial is abrupt (see Fig. 272).

Aneurysm of the descending aorta.—Many of these pass unnoticed and are revealed only by the X-rays, or at the post-mortem. In some the dorsal vertebræ become eroded. The spinal cord may be compressed. Agonizing

pain may be felt, shooting round to the front on one or both sides, accompanied by herpes zoster or hyperæsthesia due to pressure on the nerve roots. The lungs and œsophagus may be compressed. Hæmorrhage into the lungs may occur, resembling the hæmoptysis of phthisis pulmonalis. Rupture may take place into the pleura or œsophagus.

Aneurysm of the abdominal aorta.—Aneurysm may occur in any part of the abdominal aorta or of its branches within the abdomen. While of moderate size they may be recognized as pulsatile tumours. In thin persons it is easy to mistake a pulsating aorta for an aneurysm. A tumour such as occurs in carcinoma of the stomach may easily be mistaken for an aneurysm. Various expedients should be resorted to in order to be certain of the nature of the tumour, as pushing it to one side or grasping it as a whole to see if it pulsates. Another useful method is to get the patient to kneel on his elbows and knees, and observe if the tumour falls away from the aorta and ceases to pulsate.

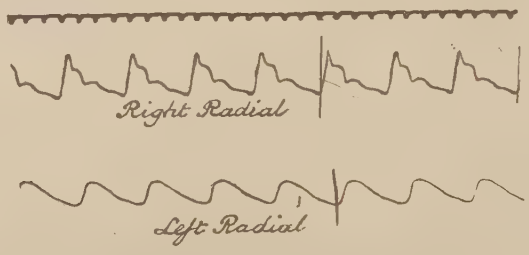


Fig. 272. Simultaneous tracings of the right and left radial pulses from a case of aneurysm of the arch of the aorta beyond the innominate, but involving the origin of the left subclavian artery. The pulses felt unequal, but the records show they were synchronous. The pulse of the right radial reaches its full height suddenly, while the left pulse reaches its height gradually. The systolic pressure in the right radial was 130 mm. Hg, in the left 110 mm. Hg.

Abdominal aneurysms tend to become very large and cause little inconvenience except from their size, but sometimes they press upon the other abdominal organs and cause erosion of the vertebræ and other bones with which they come into contact. The variety of symptoms to which they can give rise is great. When they rupture they may do so into the hollow viscera, the peritoneal cavity, the spinal cord, and so many other places that it is seldom one meets with two cases which burst in the same way.

Prognosis.—There is no rule which can be followed in all cases, as an aneurysm may result in death soon after the recognition of the condition, or the patient may live many years scarcely embarrassed. It should be borne in mind that the aneurysm may have been present for many years before its detection. In every case judgment should be suspended for a few months to see if the condition is progressive or amenable to treatment. In many cases where the aneurysm is found bulging out the chest wall, after a period of rest it may diminish in size, and the patient be able to get about his affairs and lead a useful life for an indefinite number of years,

and when he dies it is not because of his aneurysm but from some intercurrent complaint. It often happens that aneurysms of various sizes in the descending aorta are not recognized until a post-mortem examination has been made, the death not being due to the aneurysm. It must always be remembered that changes similar to those which have affected the aorta may have affected the coronary arteries, and the patient's weakness and suffering be the outcome of the cardiac condition and not of the aneurysm. Indeed, it not infrequently happens that patients with aneurysm suffer from angina pectoris, and die from cardiac exhaustion. On the detection of an aneurysm there is a tendency to attribute the danger and suffering to the aneurysm and not to the heart. When, in spite of rest, the aneurysm continues to increase in size, then the patient drifts to death, the cause of death, however, being due more often to exhaustion than to rupture.

Rupture of aneurysm is such a tragic occurrence that it is often feared after the detection of an aneurysm. It is, however, a comparatively rare event, for the great majority of aneurysms do not rupture. When they do rupture it is usually an unexpected event, and generally it is into the bronchi or lungs, and in abdominal aneurysm under the peritoneum. Some of these rupture at an early stage, before time has been afforded for the wall of the aneurysm to be supported by the consolidation of the surrounding tissues. It is therefore incumbent on the physician to watch carefully for any signs of extension after the detection of the aneurysm, either by percussion or by the X-ray. Another sign of approaching danger is blood-stained expectoration. In one case an aneurysm, revealed only by the X-ray, at the arch of the aorta on the left side, was so small in size that there was some doubt as to its nature. The sputum was, however, blood-stained, and a few days later a slight dullness to percussion indicated an increase in the size of the aneurysm, and a few hours afterwards the patient suddenly coughed up some blood and expired.

A prognosis is to be formed on the rapidity of growth and the involvement of structures necessary to life by pressure of the growing tumour. Such signs as attacks of dyspnœa and blood-stained expectoration give grave warning. On the other hand, the absence of any serious signs of pressure and the cessation of growth are, on the whole, evidences for a favourable prognosis.

Treatment.—Principles of treatment are based on the fact that while the immediate cause of the aneurysm is disease of the wall of the aorta, the heart's force is the true cause of the increase in the size of the aneurysm. The damage to the wall cannot be made good, and it is to be hoped that the wall may be strengthened by the deposit of clot on its interior. The heart's force is diminished by rest, and the formation of clot is favoured by a quiet circulation. As bodily effort increases the heart's force, it is manifest that the best line of treatment is rest, so that, as long as there is evidence of the aneurysm increasing in size, rest must be assured. When after some months there is no evidence of increase, the patient may be allowed to get

up and gradually get about his affairs, provided these do not involve too much bodily exertion.

When the aneurysm is the chief source of danger, there is often a good deal of suffering, and the pain may be taken as a guide. Thus with rest the pain disappears in most cases. When the patient gets up and goes about, so long as the pain does not return it may be taken as an evidence (in the absence of any other sign of increase in size) that matters are favourable. A return of the pain calls for a diminution or a cessation of effort.

In this matter it is always necessary to make certain that the pain and weakness are not cardiac in origin. As cardiac pain and the signs of cardiac exhaustion are very definite, little difficulty should be experienced in recognizing the purely cardiac symptom.

A great many expedients have been used to facilitate the formation of the clot and for influencing the course of disease. It is doubtful if any of these are of value. It is possible that a diminished diet may help, though there is little reason for resorting to the extreme that Tufnell and others have adopted. When it is realized that the aneurysm in many cases has existed for years before its discovery, and that many people lead useful lives for many years after it has been discovered, without any special restrictions beyond those I have indicated, it can readily be seen that cases that have been treated by some special method may do fairly well. It is, however, not justifiable to attribute the improvement to the special method unless some demonstrable result of the treatment can be shown.

Where syphilis is the cause of the aneurysm, anti-syphilitic treatment is usually recommended. But except on a general principle (of doubtful value) that all syphilitic cases should be so treated, it is difficult to see what good this kind of treatment can do. When pain is a persistent and distressing symptom, the careful administration of sedatives is called for. Certain drugs, as the iodides and arsenic, have been recommended as useful agents to prevent the growth of the aneurysm or relieve the suffering; it is doubtful if they have any effect; probably the improvement that follows rest has been credited to the drug.

Dilatation of the Aorta

As dilatation of the aorta is generally a much milder affection than aneurysm, a few words are necessary to appreciate the difference. In many people with advancing years the arteries become large and tortuous. The same processes that produce these changes in the arteries in general affect the aorta, and there is in consequence a general dilatation of the aorta. Dilatation of the aorta is also frequently present in aortic regurgitation, particularly in those cases where there is a high systolic blood-pressure and a very low diastolic. The force with which the blood is thrown into the aorta is undoubtedly the chief factor in producing this dilatation. With

extreme weakening of the walls of the aorta the dilatation may be so great that it might be considered among the aneurysms.

Symptoms of dilatation of the aorta.—The symptoms as a rule are very few, and the signs are practically limited to a slight dullness over the manubrium sterni and the detection of pulsation in the episternal notch. The X-ray is the best means for revealing its presence. It rarely produces pressure symptoms.

Prognosis.—No grave consequence is likely to result from a dilated aorta. It rarely causes trouble because of mechanical pressure. It rarely, if ever, bursts. The worst that happens is that it interferes with those peculiar features of the aorta that favour the flow of blood, chiefly by abolishing the effects of the elastic coat, and thus tends to embarrass the heart in its work, which is itself usually impaired in consequence of changes in the coronary arteries. No grave prognosis should be based upon the detection of a dilated aorta, especially in the milder degrees. This caution is needed, for its detection by the X-ray not infrequently has been the cause of unnecessary alarm.

Treatment.—As the dilated aorta is the outcome of a process that has been going on for many years, and which, in the nature of things, is irremediable, no time and energy need be wasted in attempts at treatment. If there are evidences of cardiac inefficiency, that should be the object towards which treatment should be directed.

CHAPTER LXI

The Heart in Acute Affections

Introduction.
Manner in which the heart is affected in infections.
Symptoms in myocarditis.
Symptoms in endocarditis.
Symptoms in pericarditis.
The heart in rheumatic fever.
The heart in pneumonia.
The heart in diphtheria.
Septic infections.
Prognosis.
Treatment.

Introduction.—From the description given of the manner in which the heart's contractions are carried out it was shown that the variations in rate and rhythm are dependent entirely upon the genetic system. In dealing with affections of the myocardium, of the endocardium, or the pericardium, none of the variations in rate or rhythm can be attributed to diseases of these structures. When diseases affect the heart they are rarely limited to one structure, so when we find in acute conditions such phenomena as extrasystoles, auricular fibrillation, or flutter, with evidences of myocarditis, endocarditis, or pericarditis, we can only infer that the genetic system is also affected at the same time.

The increase in the pulse-rate with the normal rhythm is not distinctive of disease of the heart itself, for an acute infection of the structures of the heart apart from the genetic system produces no more distinctive sign than the acute affections of structures in any other part of the body. This was brought out in Observations 1 and 2. It is necessary to emphasize this point, for frequently when there is a rapid action of the heart with no perceptible source of disturbance the tendency has been to attribute it to a myocarditis, or an affection of some structure of the heart other than the genetic system.

Manner in which the heart is affected in infections.—In considering the state of the circulation in febrile conditions, it is necessary to bear in mind three facts, viz. that the heart's action is modified by an increase in temperature, that the heart reacts differently according to the toxins

produced by the agent causing the fever, and finally, that the heart itself may be the seat of the conditions causing the fever. Recent researches demonstrate conclusively the invasion of the heart by the specific organism in rheumatic fever, pneumonia, typhoid fever, diphtheria, erysipelas, influenza, and various septic infections. The results of such invasion are shown in the occurrence of endocarditis, myocarditis, and pericarditis. The symptoms evoked in such invasions are not always distinctive, and may resemble the symptoms induced in the heart by febrile conditions alone, or by toxins produced from other sources in the body. I dwell upon this because an attempt should always be made in febrile conditions to judge rightly the effects on the heart. One cannot but be struck, for instance, by the fact that people with previously seriously damaged hearts may pass scatheless through severe attacks of pneumonia or typhoid fever, while the young and vigorous may succumb after a few days' illness on account of the implication of the heart in the disease.

Another point to bear in mind is that in the invasion of the heart the specific organism rarely affects one tissue alone. In order to be exact and methodical, writers usually describe separately the symptoms of endocarditis, myocarditis, and pericarditis. But if one reflects on the nature of the symptoms, such as the condition of the pulse, its strength and rate, the size of the heart, and the præcordial distress—symptoms which are usually included in the description of endocarditis and pericarditis—it will be realized that they are not really the manifestations of endocarditis or pericarditis, but are the signs of a myocardial affection. While the variations of rhythm as in Figs. 273, 274, 275 are evidences of an affection of the genetic system, one must consider carefully the murmurs arising in the course of a febrile attack, even in rheumatic fever, for the presence of a murmur does not necessarily mean the invasion of the mitral valves by the inflammatory process, but may be due to the poisoned heart muscle failing, and giving rise to incompetence of the mitral orifice—due, therefore, not to an endocardial, but to a myocardial affection, or be due to other unknown factors which produce systolic murmur. Endocarditis and pericarditis, both acute and chronic, bulk so largely in medical literature only because an abnormal sound invariably impresses the mind more than an abnormal sign perceptible by the other senses, and the easy recognition of the valvular murmur and the friction sound has led to the associated symptoms being ascribed to the same lesion.

Symptoms in myocarditis.—Evidences of myocardial affections can be found in the changes in the size of the heart. The symptoms of dilatation are described in Chapter XLVII., but it is well to remember that the size of the heart may very speedily become greatly increased in the course of a febrile affection of the heart, as in rheumatic fever, diphtheria, etc. In such cases, the sounds often become very faint, and soft murmurs may arise at the mitral and tricuspid orifices, and simulate valvular changes.

The size of the pulse being dependent on the strength of the muscle con-

traction, a feeble contraction is shown by a weak pulse, but any factors which diminish the amount of blood also produce a weak pulse. The rate of the pulse is affected in the same way as it is in inflammation of any other part of the body.

Symptoms in endocarditis.—The only direct evidence of acute endocarditis is the presence of murmurs at one or other orifice of the heart. For practical purposes there are only two murmurs which need to be considered here, viz. a mitral systolic murmur and an aortic diastolic. It is

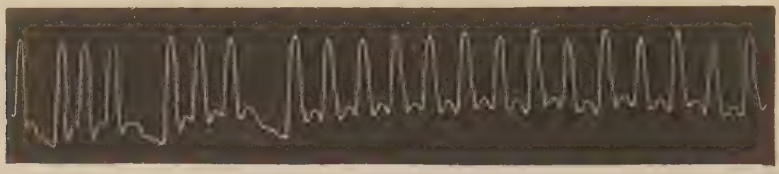


Fig. 273. Irregular pulse in the course of a pneumonia, showing the pulsus alternans in the latter half of the tracing.

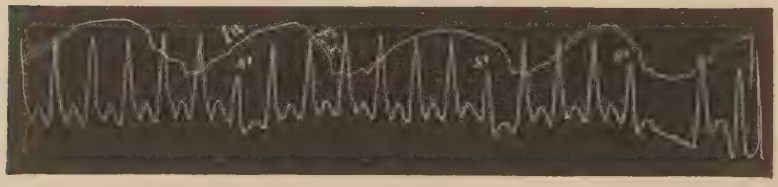


Fig. 274. Tracing of the respiratory curve and of the irregular pulse in the course of a fatal case of pneumonia.

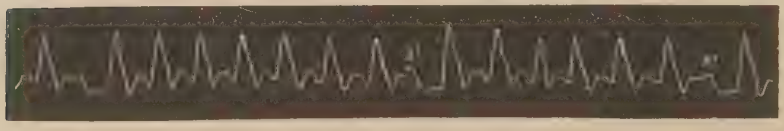


Fig. 275. Extra-systoles occurring in the course of a fatal attack of rheumatic fever.

not always easy to tell whether the appearance of a murmur during a febrile attack is due to involvement of the mitral valve in an endocarditic process, or to the relaxation of the muscle supporting the orifice. A diastolic aortic murmur is, as a rule, diagnostic of the involvement of the aortic valve in some destructive process. At first this murmur is so faint that one is simply conscious of the fact that the sound does not end with sufficient abruptness. Gradually, however, this passes into a very soft short whiff at the end of the second sound, becoming day by day more marked.

In the vast majority of cases the murmurs due to endocarditis are not definitely recognized until some time after the subsidence of the fever—

when the cicatrizing process sets in. This is particularly the case with presystolic mitral murmurs, which do not occur during the acute condition that induces the lesion, unless there is narrowing of the mitral orifice on account of vegetations. The formation of vegetations at the mitral and aortic orifices may give rise to murmurs, indistinguishable from those due to destruction of the cusps. The presence of a musical murmur may generally be assumed, particularly in acute cases, to be due to a vegetation. An attack of hemiplegia during an acute febrile condition may generally be ascribed to an infarct from a valvular vegetation, and infarcts in any other organ may be assumed to arise from the same cause.

Symptoms in pericarditis. — Until the introduction of auscultation, dry pericarditis was a disease only discovered on the post-mortem table. The only evidence we have of its presence is the characteristic superficial to-and-fro murmur produced by the movements of the heart. Its discovery is usually accidental, and made when the heart is examined as a matter of routine. There is no other distinctive sign associated with it, and, in marked contrast to dry pleurisy, it is essentially a painless complaint. When pains are associated with its presence, it will invariably be found that there is evidence of a myocardial affection. This curious painlessness of pericarditis compared with pleurisy is one that has long puzzled me, and I have only a dim perception of how it may arise. Some writers describe pain as a symptom of pericarditis. It is possible that a dull ache may be present, but I feel fairly certain that when there is a severe pain aggravated by a movement of respiration the pain is due to a coincident pleurisy.

Pericarditis may arise in the course of a number of chronic complaints, as in diabetes and Bright's disease, or in the course of an acute disease, as pneumonia or rheumatic fever. Sometimes one comes across it quite accidentally—the patient, not feeling quite well, consults his doctor, and in the course of an examination this is detected. Such patients may go quietly about their occupation for many weeks with a well-marked to-and-fro murmur, and suffer no further trouble.

When effusion takes place into the pericardial sac, there is an increase in the area of the cardiac dullness, varying with the amount of fluid. It is difficult to recognize a small amount of fluid, particularly if the heart is enlarged. When the amount is considerable the area of dullness assumes a somewhat characteristic shape. It reaches up to or above the second rib, and if the area be mapped out, it will have a somewhat pear-shaped character. There is an absence of the heart's movements at the lower point to the left, and this should always arouse the suspicion of effusion where there is an increase in the heart's dullness. Ewart describes a small area of dullness behind at the base of the left lung. This is important to remember, for an increase in the size of this area may cause an extensive pericardial effusion to simulate fluid in the pleural cavity. I have tapped a pericardial purulent effusion in mistake for an empyema, and my mistake arose from not ascertaining the position of the heart's movements. Had the case been one of

pleural effusion, I should have found the heart beating to the right of the sternum; but the whole left chest was dull, so that the idea of it being pericardial never crossed my mind.

The question of pericardial effusion embarrassing the work of the heart has arisen on account of the results of experimentally distending the sac with fluid. I have never found serious embarrassment of the heart from extensive pericardial effusion, the reason being probably that while the normal pericardium is a more or less inelastic bag, with the inflammatory invasion it becomes distensible, and therefore able to accommodate an enormous amount of fluid with little embarrassment to the heart. I have seen enormous effusions into the pericardial sac from various causes—as pericarditis, purulent pericarditis, blood-stained serum, due to a neighbouring cancer—and I could not see any bad effect on the heart's action. I have tapped and drawn off large quantities of fluid with no benefit to the patient. Still my experience may be too limited to enable me to speak dogmatically on the subject.

The heart in rheumatic fever.—The real nature of the changes that take place in acute affections of the heart is still far from clear; but it is possible to connect some of the obscure signs observed during life with the disease process in the heart. The changes which occur in rheumatic fever occur also in other acute affections, as in pneumonia, diphtheria, influenza, septic poisoning.

The heart trouble starts with the invasion of the heart by an organism. The invasion of the endocardium usually affects first the base of the valves, and produces swelling and infiltration of the margins. The swollen edges may break down and ulcerate, or vegetations may form. The course of the disease varies greatly, from the simple endocarditis which recovers, to extensive ulceration of the valves, associated with the severe symptoms characteristic of malignant endocarditis.

The myocardium rarely escapes, and the changes in it are of great importance, both for the acute condition and for the subsequent integrity of the heart muscle. Fatty degeneration and breaking down of the muscle-fibres are fairly common, while the specific organism has been found accompanied by cellular infiltration. There may be congestion of the blood-vessels, exudation of the leucocytes, and swelling of the connective tissues. Aschoff describes the occurrence of numerous cellular foci.

During the acute attack extreme dilatation may occur, due probably to a toxæmic poisoning of the heart muscle, as no microscopic change may be present. This poisoning is probably also the cause of the extreme weakness of the heart, which persists for some time after the subsidence of the fever. The pericardium is also liable to invasion, and here the changes may vary from a slight transient pericarditis to an extreme inflammation, which does not entirely subside, but lingers on, forming adhesions to the tissue outside the pericardium and penetrating to the heart itself.

A process of slow cicatrization often follows, producing changes in the

valves, heart muscle, and pericardium that seriously embarrass the heart in its work in after years.

Symptoms.—Attacks of rheumatic fever may complete their course with no affection of the heart. In some instances, the heart may be affected and give rise to no positive sign ; and it may only be after months or years that a murmur or irregular action indicates that there must have been some affection of the heart which the symptoms produced by the subsequent cicatricial process have revealed.

Generally, however, we can recognize certain changes in the heart's condition, chiefly an increase in the size and the presence of a murmur. These cardiac changes may go on with very little increase of temperature, and little or no evidence of joint trouble. Sometimes, in these milder cases, there is evidence of involvement of the auriculo-ventricular bundle producing partial heart-block (Observation 63), the significance of this observation being that it is a sign that the myocardium has been invaded by the disease and damaged. The accompanying damage may be slight, and give rise to no sign, or there may be extreme damage, revealed by an increase in the size of the heart and its functional impairment.

With more serious involvement the dilatation of the heart may be extreme, and it may be mistaken for pericardial effusion. At first, the full extent of the enlargement may not be realized, because it is partly masked by the lung. When the lung is pushed aside the greatly enlarged heart can then be readily recognized. The rate of the heart is usually greatly increased, beyond what might be expected from a mere rise in temperature. The pulse becomes soft and compressible, and sometimes shows irregularities whose nature in all cases it is not easy to make out. With subsidence of the fever, the patient enters on a long and slow convalescence. Other cases do not terminate so favourably, especially if the heart has been damaged by a previous attack. Complications, as pneumonia, are apt to arise. In severe cases there may be a considerable amount of præcordial distress. The breathing becomes shallow and rapid. The patient feels easiest with his shoulders well raised. The face becomes dusky, the lips dark red, sleep is broken and fitful, and the patient is continuously altering his position. The mind wanders and mental delusions arise.

From such a state as this the young during their first attack may recover, but in the middle-aged the condition is very serious. Attacks of syncope may appear, and the patient may die in one. Frequently they gradually sink, in spite of all treatment, and die.

In the recurring attacks of rheumatic fever, this question of previous damage to the heart is a very important one. Patients with damaged aortic and mitral valves may pass scatheless through serious attacks of rheumatic fever, presumably where the heart is not involved in these later attacks. When, however, the process lays hold on the heart, the patient's life is in great danger, and after a period of extreme suffering the struggle frequently ends in death.

While the foregoing description gives briefly the main points of the heart affection in rheumatic fever, it also holds good for the condition in other infectious diseases, apart from the recurrent attacks. As, however, the presence of other lesions has a modifying effect upon the course of the disease, it is necessary to refer to them. Unfortunately, the reference can only be brief and in the main unsatisfactory, the analysis of the symptoms in these cases having been very imperfectly carried out.

The heart in pneumonia.—The invading organism may assail the heart as well as the lungs, and the course of the illness be rapid and severe. It is difficult to distinguish between the changes due to invasion of the heart and those due to the general infection. In severe cases the evidence of the heart affection is very prominent. Within a few hours of the initial rigor, and before there is any pulmonary sign, the evidence of the heart affection is all too apparent. The patient may be young and, prior to the attack, a perfect specimen of youthful health and vigour. A few hours after the rigor, the temperature may be over 102° F. The condition of the pulse is the best guide to the state of the heart at this stage, and shows ominous signs of what is to follow. It is increased in rate, 115-130 per minute. It is soft and compressible, and offers no resistance between the beats. The peculiar manner in which it impinges against the finger—sharp and short, then quickly subsiding, indicating an absence of sustained pressure—is always an ominous sign. It is usually associated with greatly relaxed arteries, and a sphygmogram shows little or no sign of a dicrotic wave, indicating great lowering of the pressure during the diastole of the heart. The heart itself shows no definite sign. The sounds are short and sharp at first, becoming later somewhat muffled. A certain amount of dilatation occurs, detected more particularly to the right of the sternum. Usually in these cases the end comes with tragic suddenness; the rate of the pulse increases, irregularities appear, and the patient succumbs within three or four days of the initial rigor. Figs. 276-280 show the characteristic features of the pulse in acute fatal pneumonia in a young, previously strong and healthy adult.

The heart in diphtheria.—The complications here are so varied that danger may arise in several quarters. The heart muscle itself may be the seat of profound changes, the symptoms somewhat resembling those in rheumatic fever. But in diphtheria, more than in any other acute disease, there is a tendency to fatal syncope, and I do not understand how this is brought about.

Septic infections.—There are a great many septic infections that injure the heart, either from toxæmia or by a specific organism invading the heart. In the latter case, the endocardium is frequently attacked, and the disease is then described as septic endocarditis. In these cases there is also invasion of the myocardium, and it is the profound depression of the heart muscle which is often the grave element.

In an account of 150 cases of infective endocarditis, Horder states that

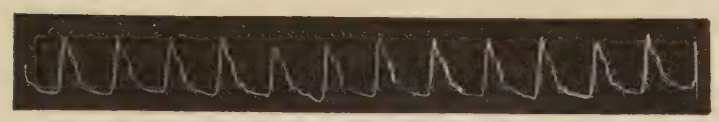


Fig. 276. Febrile pulse of low arterial pressure, T. 103°, P. 116, R. 36. This was taken eight hours after the rigor at the beginning of a pneumonia. This and the four following tracings show a type of asthenic pulse.

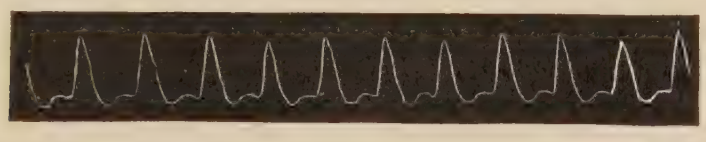


Fig. 277. T. 101·5°, P. 96, R. 28—second day.

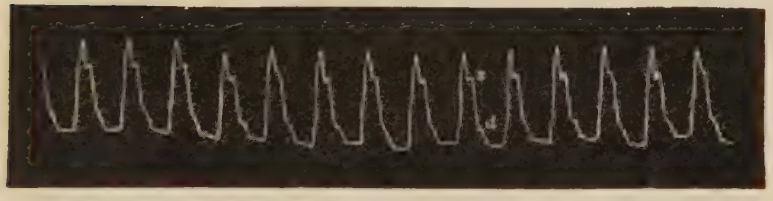


Fig. 278. Asthenic type of pulse with well-marked systolic wave (*s*), and only a faint indication of the dicrotic wave (*d*)—third day.

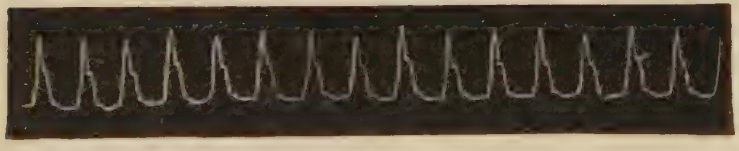


Fig. 279. T. 103°, P. 124, R. 48—fourth day.

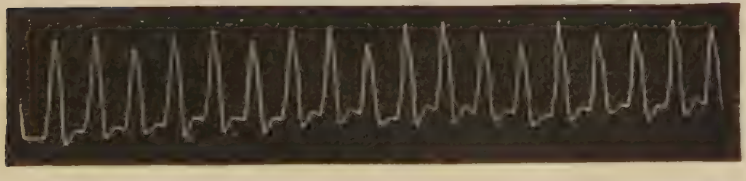


Fig. 280. T. 102°, P. 148, R. 52. The irregularity and rapidity of the pulse heralded the fatal end on the fifth day.

in 90 per cent of cases a culture of a pathogenic organism can be obtained. The following table gives the analysis of forty positive results of blood culture during life :—

Number of cases.	Micro-organism isolated.
26	Streptococcus.
5	Bacillus influenzæ.
5	Pneumococcus.
2	Gonococcus.
1	Staphylococcus albus.
1	Unclassified.

The illness in these cases usually begins insidiously, and at first may be mistaken for some trivial febrile complaint, or for influenza. Soon, however, the extreme prostration of the patient, the recurrence of rigors, and the patient's own sensation of illness, show that the condition is of a more serious nature. Usually, also, there is excessive perspiration. If the heart be watched, it will be found to dilate and a systolic murmur appear. The true nature of the condition may not be revealed until the detachment of a vegetation produces hemiplegia, or an infarct in the spleen or kidney or elsewhere, and death may speedily supervene (malignant endocarditis).

Other cases may linger on with indefinite febrile attacks, the patient sick, pale, and ill, and the real nature of the illness be a mystery. In some of these cases very little change takes place in the heart. I have seen a case after confinement have slight fever for nine weeks with no change in the size of the heart, the rate generally about 80 per minute, a rough systolic mitral murmur the only abnormal sign, until an attack of hemiplegia led to the recognition that the rough mitral murmur was due to vegetations on the valves. In addition to the irregular fever, Osler gives the following as the most suggestive features which help to identify the nature of the disease : (1) A knowledge of the existence of an old valve trouble ; (2) the occurrence of embolic features, sudden swelling of the spleen, sudden attack of hæmaturia, embolism of the retinal arteries, hemiplegia, or the blocking of a vessel in one of the limbs ; (3) the onset of special skin symptoms, purpura, and more particularly the painful erythematous nodules, in all probability due to minute emboli ; (4) the progressive cardiac changes, the gradual increase in the dilatation of the heart, the marked change in the character of the mitral murmur, the onset of a loud rasping tricuspid murmur, or the development under observation of an aortic diastolic bruit. During the war many patients obviously ill gradually developed symptoms similar to those, which led to the recognition of the disease as fairly frequent. In them there was often no previous history of a damaged valve.

In infections like ' malignant endocarditis ' and puerperal septicæmia, we get conditions of profound gravity caused by certain organisms. The pulse in these cases gives the most trustworthy information. It is small, soft, and easily compressed, not necessarily very rapid, and the temperature need not be high (101-2°). The heart shows little change except that the sounds are

feeble; the patient is lethargic, the face is slightly sallow or pale and sunken. The aspect of the patient, with the condition of the pulse, affords assistance in recognizing the condition. These signs are probably due to dilatation of the capillaries absorbing most of the blood. In the post-mortem examination little or no blood is found in the heart or large veins (see p. 79).

Prognosis.—In acute disease due to some infective agent, the prognosis depends upon the nature and intensity of the infection. So far as the heart is concerned, judgment must be suspended till the cessation of the active lesion. As these effects may linger on for some time, it is manifestly impossible to do more than lay down certain general rules. After acute affections, such as rheumatic fever, and after subacute affections, no prognosis should be given until all the symptoms of the original affection have disappeared. If the heart has resumed its normal aspect, in size, rate, and rhythm, we may gather that the damage has been slight and that the future is hopeful. The presence of the youthful or respiratory irregularity may be a further guarantee of the integrity of the myocardium (see Chapter XXII.). If some evidence of damage persists, then we must recognize this, lest it be due to the persistence of some low inflammatory condition which leads to the formation of fibrous changes, in which case it may be a long time before the changes are sufficiently stationary to permit of an opinion.

The prognosis of an infective endocarditis apart from that due to rheumatic fever is usually very grave. The condition often escapes recognition until some advanced sign occurs, as an embolism.

Treatment.—When the real nature of the trouble is appreciated in acute affections of the heart, it will be realized how powerless we are directly to modify the disease process.

In rheumatic fever the salicylates have an undoubted action on the course of the disease in many cases, and it may be that the drug can modify the heart affection. Their employment is so frequently of use that it should always be tried, and given in such doses that a physiological reaction is obtained.

Apart from the probably specific action of salicylates in rheumatic cases, the employment of cardiac or other drugs is of little avail.[1] The heart is already assailed by a poison far more powerful than the drugs at our command, and these in medicinal doses are without effect. But on this account it must not be supposed that all treatment is useless; rather should our attention be directed to the consideration of other means of treatment. The

[1] Horder says: '"Blood antiseptics" seem doomed to failure in dealing with pyogenic blood infections, because it is not possible to get the drugs into the blood in a nascent or active condition. Combination with the proteids of the blood-cells or the plasma takes place before the drug comes into contact with the micro-organism. Quinine, mercury, arsenic, carbolic acid, formalin, and many other reputed remedies, all fail, whether administered by the mouth, subcutaneously, or intravenously. The sulpho-carbolates, which have had a special vogue, are equally disappointing; I have used them in very large doses in several cases without any results. Silver salts in combination with nuclein have been highly spoken of in the treatment of septicæmia, but here again I have never seen any good results in infective endocarditis. The same remarks apply to yeast and its active principle.'

man who puts his faith in drugs exclusively neglects too often the most useful methods. Recognizing that the heart muscle is greatly embarrassed in its work, the endeavour should be made to give it as little work to do as possible, and to save it from all sources of irritation ; in fact, to place it in a condition of rest, as far as rest is possible for such an active organ as the heart. To this end the general condition of the patient should be carefully studied ; the position he assumes should be one that gives the heart least work to do ; the food should be so administered that while nourishing him it does not lead to abdominal distension : his bowels should be so regulated as to act freely without straining. As fidgeting and restlessness keep the heart irritable and variable in its action, everything should be done for his bodily comfort—sponging, and arranging his pillows and the bedclothes, and the many little things that a deft and intelligent nurse can suggest. Above all, sleeplessness, which is so often present, or the sleep that is broken and disturbed, should have most careful consideration, and if necessary suitable hypnotics be given.

When there is reason to suspect that the heart muscle has been affected by the illness, great care should be taken during the convalescence to give the heart muscle time to recover. Any cause, effort or excitement, that accelerates the heart's action, should be avoided, and exertion only permitted when the dilatation has subsided, and effort ceases to call forth any disagreeable sensation. It may be weeks or months after the fever before the heart muscle recovers.

CHAPTER LXII

The Principles of Pathology and of Treatment

Pathology

In my student days it was dinned into us that pathology was the foundation on which medicine should be built. At that time new methods were being introduced for the examination of the tissues by staining microscopical specimens. Such specimens were studied with the greatest particularity, and it was thought at that time that the cause of ill-health and of death were to be revealed by the examination of morbid specimens. That notion prevails still, for we find post-mortem examinations being made in the expectation that the cause of death will be found.

As years pass on the expectations of what pathology was to do have not been fulfilled. Valuable as research into post-mortem conditions has been, and helpful as it has proved in the investigation of disease, it does not reveal the kind of knowledge which is wanted. All that it reveals is damaged structure. Ill-health and death are brought about by interference with function, and disturbed function cannot be revealed by the investigation of dead structures.

What is wanted is a functional pathology, that is, a knowledge of disturbed function. That is, no doubt, a trite saying which every one will accept, and in recent years, institutes of pathology have been organized for the purpose of studying pathological processes in the living, but it is difficult to understand how such investigations can help medicine without a knowledge of functional disturbances. These are manifested only by certain unusual or abnormal reactions, that is, by symptoms, and it seems perfectly plain that before one can study the nature of the functional disturbances one must get a knowledge of the manner in which symptoms are produced. If this knowledge is not acquired then the interpretation of the reactions which the investigator may produce can only be a matter of conjecture, and the methods by which nature produces its effects can never be revealed by conjecture.

So far as I can see, pathology follows the custom of modern investigations to which I have already called attention, of detecting end-results and guessing at the manner of their production (pp. 36 and 37).

In this book an attempt has been made to approach pathology from a rational point of view. I do not pretend for one moment that much progress

has been made, but the few steps that have been taken clearly indicate the line for future progress as they tend to reveal the disturbances of function with a precision and accuracy not attained by any other method. This was shown in Chapter VIII. and in other places where the principle of the reflex arc was applied.

From the descriptions given in this book, it has been shown that the symptoms of disease are the result of agents which act temporarily or are the result of organic changes. These agents only modify the rate at which the energy of cells is restored after they have discharged their function. This was illustrated in the production of partial heart-block, where the agents of disease delayed the restoration of energy, and acted in the same manner as a drug, like digitalis. Belonging to this category are all those increases in the rate of the normal acting heart in various febrile states, where the agents of disease hasten the restoration of energy.

Identical results are produced in many instances by changes of structure, and these changes are so little understood that the innumerable manifestations that they give rise to cannot be classified on that basis of their affinity in nature which is necessary in dealing with natural phenomena. For many years this has been an object of research on my part—approaching the subject by means of the opportunities offered to the general practitioner. The best method was by observing and interpreting the changes which accompany advancing years. The post-mortem study of these changes does not give the kind of knowledge required for their understanding, for the changes have usually advanced so far that the primary lesions are lost in the gross changes which result. It was quite evident that old age meant the impairment of the functional activity of organs. This impairment may be shown most prominently by one organ in one man, and by other organs in other men, while in some men the impairment of function is exhibited by more organs than one. Moreover, out of several men in whom the same organ was affected, each showed different symptoms.

The custom of medicine hitherto has been to describe the impaired functions as due to a particular form of disease, and it is on this basis that most text-books dealing with general medicine are written. In this way diseases are described according to the organ which exhibits them, and not only according to the organ, but according to the part of the organ which may be affected. Thus it comes that pathological states are so numerous as to be difficult of comprehension.

Looking at it from the point of view of the general practitioner, the conclusion was come to that in the vast majority of individuals there must be one common factor, and that the infinite variety of manifestations was simply due to the difference in the structure which was affected, and in the function which was disturbed.

In looking for this common factor, I found it to be due to an insufficient supply of blood. Although the leading symptoms and disabilities could be attributed to one organ, such as the heart, the brain, the kidney, the stomach,

the legs, etc., yet there were invariably present distinct evidences of changes in other organs. The most apparent of these was in the skin. In practically every instance it becomes thin and attenuated in certain parts, as in the back of the hands, and the front of the legs. In operating in places where it was not so evident, as in the abdomen, bleeding from a cut was not of that character found in the young, where there is a general oozing from the cut capillaries. Frequently the bleeding was only from the small vein, or a small artery with thickened walls.

From such evidence the question arose, 'Was not this obliteration of the capillaries the real source of all the degenerative changes that took place?' and this aspect of the subject has been dealt with in Chapter XIV.

In our researches at St. Andrews we paid particular attention to this aspect of the subject, more particularly giving consideration to the eye and to the heart.

The results obtained by the examination of the heart may be regarded as typical of every other organ, so that the nature of the heart trouble which occurs in advancing years depends on the structure in which the capillary obliteration is most advanced. In this way such diverse diseases as valvular disease, angina pectoris, auricular fibrillation, and heart-block are all seen to be due to the same morbid process. In some cases we were successful in recognizing how one diseased state merged into the other. We found this, for instance, in one man who recognized his limitations at the age of 74, because of pain across his chest on exertion. On examination there was found an aortic systolic murmur. A year later he began to have attacks of loss of consciousness, which were found to be due to partial heart-block. This block ultimately became complete, and he suffered no longer from pain on effort, but from weakness and a sensation of constriction across the lower part of the chest. He died suddenly at the age of 77. At the post-mortem examination certain definite changes were detected, from which, with his history, it is possible to reconstruct the progress of the disease. There had been gradual changes taking place in the aortic valves of a sclerotic character. The sclerosis had extended up the aorta and embraced the mouth of the coronary artery so that it was narrow and rigid and incapable of expanding to permit of a larger supply of blood during effort, with the result that pain occurred on making an effort. The process had extended downward to the a.-v. bundle, which it invaded and destroyed, so that partial, then complete heart-block followed. Clinically, therefore, his condition could be diagnosed as Disease of the Aortic Valves, Angina Pectoris, and Heart-Block, but pathologically, there was but one disease process.

The series of hearts which are represented in Plates I.-IV. are given to illustrate advanced diseases of the heart which came about through gradual obliteration of the capillaries that supplied the walls of the coronary arteries.

In Observation 62, p. 289, there is given an account of a man where

the gradual progress of the process affecting the aortic valves was revealed by the alterations in the sounds.

Reference has also been made to this point of view in Chapter XIV.

The presentation of the subject in this way is to show that a great variety of so-called diseases are produced by one morbid process. The various diseases of the kidney, the brain, the digestive system, and such diseases as the glycosuria of advanced years, are indicative of a diminished blood-supply. The disturbances of function which result are not peculiar to this morbid process, but are the same for other morbid processes, such as new growth, and microbic invasion—just as the symptoms of angina pectoris are the same for both rapid advancing syphilitic affections and slow progressing sclerotic changes.

By this method of looking at disease we are able to look at individual cases with clearer vision. The occurrence of auricular fibrillation reveals the presence of disease changes. The patient's future health has been shown to depend, not upon the fibrillation of the auricle, but on the extent of changes that may have occurred in the ventricle—so that it is the doctor's duty to find out if there are morbid changes present.

This view was discussed under Prognosis (p. 21), and shows that such diagnosis as 'valve disease,' 'auricular fibrillation,' when heart failure is present, is imperfect and unsatisfactory, and reveals the need for a form of pathology in which the clinical evidences are recognized and correctly interpreted.

Treatment

These observations show that for a diagnosis a knowledge of the pathology of function, as well as of structure is necessary. They also show that treatment can never be rational until the nature of the disturbance which produces ill-health is recognized. For this purpose it is necessary to assess the value of any sign or symptom that has been detected. Hitherto this has been very imperfectly done, but the principle of the reflex arc when applied to the interpretation of the sign or symptom, discloses whether it is due to a structure upon which the integrity of the heart's functions depends, and in this way, this principle guides to that kind of knowledge which enables the doctor to understand whether there is danger present, the source of the danger, and the steps to be taken to combat the danger, if this is possible.

Once the significance of the assessment of the value of symptoms is recognized it will be seen how treatment is still governed entirely by empiricism ; and the urgent need, if the practice of medicine is to make progress, that steps should be taken to obtain a truly scientific therapy. If we define empiricism as the giving of a remedy whose action is unknown for a condition whose nature is also unknown, it will be seen that it accurately describes the treatment of the present day. The origin of treatment goes back into the dark ages when it was found that certain agents introduced into the body produced demonstrable effects. From that time continual trials have

been made of every form of agent that the ingenuity of man at all stages of civilization could devise. What is, in recent years, called the 'scientific therapy' is but a continuation of the same empiric method. The ingenuity of laboratory workers has extracted from animals various agents such as extracts from the organs of the body, vaccines, and sera. Such agents may have demonstrable effects, some of them beneficial, others the reverse. Still the method pursued is the same, nobody having a knowledge of how the remedies act, or a knowledge of the conditions for which they are given.

It is in this field where the need is so urgent for the application of the principle of the reflex arc. It compels a diligent search for the structures and processes which produce the ill-health. The recognition of these gives a specific aim for the application of a remedy. As the remedies themselves act in the same way as the agents of disease, the application of this principle guides to an understanding of how the remedies act.

The progress we have made in the application of the principle in treatment is very slight. There is no prospect of much progress for a long time. The kind of knowledge required is not to be had except in a few instances. Before the investigator into the action of remedies can hope to make any real progress he must learn a great deal more about the manner in which organs function. This knowledge is to-day not available and treatment is bound for a long time to remain empiric.

While so far little has been done, yet that little clearly shows what possibilities lie open to those who have grasped the meaning of this principle. An enormous amount of time and energy has been wasted by hundreds and even thousands of people in investigating the action of digitalis, simply because of the absence of a principle to guide them. For over one hundred and fifty years digitalis has been recognized to be a potent agent in certain affections of the heart, and to be impotent in numerous other affections. The differences in the response to the drug had baffled all attempts at explanation. The reason why its action was impossible to discover, was the fact that the structures and the functions of the structures upon which the drug acted were unknown. Innumerable laboratory experiments have been carried out, but they throw no light upon the problem.

This is the point which it is necessary to grasp, for it demonstrates so clearly the inadequacy of modern conceptions to solve the problems of medicine. Whenever there is an inquiry into the action of a remedy such as chloroform, or digitalis, the matter is handed over to the individual whose opportunities have rendered it impossible for him to acquire the necessary knowledge.

If there is one thing that has come clearly out in this inquiry more than another, it has been that digitalis acts more powerfully on certain abnormal hearts than it does on normal hearts. So much so is this the case that one can formulate the proposition that the action of digitalis on the human heart is modified by the disease that may be present. If evidences are sought for it will be found that this proposition is of universal application.

If the grounds on which such a principle is based be sought, it will be found

that the agents of disease act in exactly the same way as therapeutic agents, whether they be drugs, gland extracts, vaccines, or sera, so that the action of remedies which affect sick people can never be discovered by the study of their effects on healthy individuals.

This does not imply that experimental observations are valueless. Far otherwise. The testing of a remedy upon a healthy animal may give an indication upon what structures it acts. But before this can be done, a knowledge of the structures and processes concerned in the reactions of organs must be acquired, and such knowledge can only be obtained by clinical investigation.

The evidences for this contention are given in this book. It has been shown that the hearts of patients with auricular fibrillation and flutter react in a totally different manner from normal hearts. It was shown that the reason of this was that morbid conditions had resulted in causing the ventricular contraction to be under the influence of the a.-v. node, and it is this node which, in auricular fibrillation and flutter, governs the activity of the ventricle. When the excitability of the a.-v. node is raised there results a great increase in the rate of the ventricle, with numerous small imperfect beats, and as a consequence, heart failure.

The definite object in such a case is to diminish the rate of the ventricle and increase the strength of the beats. It is shown that this can be achieved by digitalis, and thus we have before us not only a specific object for our treatment, but a recognition of the structures on which the drug acts, and how it does so.

But there is still much to learn. The question arises, 'Does the drug act directly upon the node, reaching it by way of the blood stream, or is it through the action of the vagus ?' The numerous facts to be found in clinical medicine indicate that a vagal stimulation does frequently have a marked effect upon the a.-v. node, and there are therefore many reasons for attributing this beneficial effect to the action of digitalis upon the vagus.

Then there is the further matter to be considered, 'What portion of the vagus is acted upon ?' If we look at Fig. 10, which may be taken to represent a vagus neurone, there are four parts concerned in its activity, the receptor fibrils, the discharging fibrils, the conducting fibre, and the cell body. The conclusion arrived at at present is that digitalis acts upon the receptor fibrils, raising their excitability. For this view such reasons are found as the fact that when digitalis is found to affect the heart it also at the same time produces other effects such as nausea and vomiting. Again, in experiment, when an animal has been digitalized and its vagus nerve cut, its effect is at once lost.

Atropine is recognized as paralysing the vagus by acting upon the discharging fibrils so that the one drug acts upon the receptor fibrils and the other on the discharging fibrils.

The value of having a well-defined object is seen in heart-block. The independent bundle has been shown to be very unexcitable, so much so that it may fail to send impulses to the ventricle and death may ensue. Just as

there was a definite object to aim at in the treatment of auricular fibrillation, viz. to reduce the excitability of the a.-v. node, so here there is a definite object, viz. to find a remedy that will increase the excitability of the bundle.

In such considerations it is necessary to remember the nature of the structure with which we are dealing. It is for that reason that so much attention has been directed to the genetic system.

In seeking for a principle in treatment the pathology of the condition has to be considered in all cases. In primary angina pectoris the principle of treatment becomes readily recognizable. The structural changes being irremediable the treatment then is devoted to saving a diminishing number of muscle cells from over-activity. This requires a careful study to find out that amount of effort which can be safely undertaken, and the obtaining of this knowledge gives a definite object for investigation, the results of which were described in dealing with the treatment of this condition.

The object of dealing with these matters is merely to indicate the lines on which a scientific, as distinct from an empiric therapy, should be based. Whatever may be said to the contrary, no steps of any value have hitherto been taken to obtain a scientific therapy. Nor could such steps be taken until the principle of the reflex arc was applied. There seems little doubt that until the kind of knowledge which the application of this principle reveals is acquired it is hopeless to look for a scientific therapy.

It has been shown that the reactions which we recognize as the symptoms of disease are the outcome of agents that act temporarily or are due to permanent structural changes. If the former, the line of treatment is to remove the agent or to combat the deleterious effects it produces. If the latter the object should be to make the best of a damaged organ.

The abnormal rhythm of the heart, like auricular fibrillation, may be due to either condition, and the line of treatment to be pursued depends on the recognition of the condition. If the former, the attempt may be made to restore the normal rhythm by the use of quinidine ; if the latter, then the line of treatment will be directed to preventing the rate of the heart becoming too frequent.

It is the same in angina pectoris. If it is the secondary, the treatment should be directed to removing the causes of the ill-health of which the angina is but one of the evidences. If due to organic changes, then the line of treatment is to conserve the impaired heart.

I know these are simple self-evident propositions, but it is the simplest things in medicine that are overlooked. Some people imagine that every case of auricular fibrillation has to be treated either with digitalis or quinidine, while the forms of angina are so little differentiated, that the innocent and the grave cannot be distinguished, and we see to-day surgical treatment employed without any discrimination whether or not the patient is likely to be injured by the operation.

Pharmacological research is sometimes regarded as being of little profit, and sometimes the interpretation of its results may be misleading. But this

form of research is, like so much in medicine, the application of a useful method to purposes for which it is unsuited. If it was realised that the reactions of drugs resemble the reactions produced by the agents of disease, its employment would undoubtedly throw light upon many of the problems of disease. Moreover, as the agents used affect the activity of natural forces, it would afford a far more accurate knowledge of the manner in which organs carry on their functions than that employed by the use of electricity as a stimulating agent. In the former instances natural reactions are produced, and in the latter the reactions are artificial. There is an important field for investigation in pharmacology—after the knowledge is acquired of the processes and structures that are engaged in modifying the activity of organs.

CHAPTER LXIII

The Examination of the Patient

Methods of examination.
The sense of exhaustion and fainting.
Exhaustion in ill-health.
Differentiation.
The mechanism of exhaustion.
The clinical significance of the sense of exhaustion.
Exhaustion due to an ineffective heart-beat.
Exhaustion due to vaso-motor disturbances.
Exhaustion and the response to effort.
Fainting.

Methods of Examination

In the examination of patients different methods have to be adopted according to the nature of the patient's complaint. The doctor in his examination should, at an early stage, recognize the main object to which he should direct his attention. When this object is concerned with the circulatory system, a definite line of procedure should be followed. His observations should begin from the moment his eyes rest upon the patient, taking a mental note of such matters as the manner in which the patient walks into the consulting-room or the position assumed, if in bed; the presence or absence of respiratory distress, the colour of the face, the presence of lividity of the lips, of the cheeks, of the ears, the presence of a pallor with or without a tinge of yellow, or a pallor associated with a shrunken appearance of countenance and manifest torpidity of the patient; the flushed countenance and other indications of increased vascularity as well as the more apparent signs of varying degrees of jaundice. These and many other observations that the trained eye can in a moment of time detect should be memorized and used to direct the further examination. These observations alone may give the essential information on which a diagnosis and a prognosis can be made.

The next step after taking the patient's name, age, and occupation is to obtain a clear and succinct account of the sensations by which the ill-health was recognized, or other reasons which influenced him to consult the doctor. In obtaining this account the patient must be kept strictly to the matter in hand, and not allowed to introduce irrelevant matter, and all tendency to garrulity must be firmly suppressed.

This is to be done by the doctor having in mind very clearly the kind of information he wants. He must therefore frame his question in a way suited to his patient's comprehension. He must see that the answer is complete, and must not inquire into any other matter until satisfied on this point.

The case being one of suspected heart or circulatory disturbance, the questions to be asked become very definite. The essential matter is the presence or absence of failure of the circulation, and particularly of the heart.

There is only one way to discover this, if the patient is able to be up and about, that is, to appreciate the response to effort. The questions, therefore, should be directed to the specific purpose of finding this out. The patient should be asked how he comports himself when at his very best, in walking on the level or up hills. Then, if there is any limitation, the question should be asked, what is the sensation which warned him that he had done enough, or which prevented him going farther. If the sensation is other than breathlessness, or pain in the chest and arms, or in the neck, the probabilities are that the reason for his limitation is not cardiac in origin (see p. 414). If it is due to exhaustion, then the line of inquiry should proceed on the assumption that exhaustion is secondary to some other condition, probably of a toxic or infectious nature.

It may be that certain questions do not disclose such symptoms. Then the doctor must proceed to inquire if other circumstances may provoke such signs as breathlessness and pain, such as effort on a cold day, or walking against a wind, or after meals, or in passing from a warm room into cold air. Also whether the patient can sleep comfortably with one pillow, or if there is distressed breathing so that the patient has to be propped up.

In the absence of any of these signs of limitation, and with a response to effort such as experience indicates to be that of a healthy individual of the same age, one can draw the conclusion that the functional efficiency of the heart is good.

The distress of the patient, however, may come on at irregular intervals and last for a period. The probabilities then are that there is some other circumstance tending to impair the efficiency of the heart. These circumstances should be inquired into, and they may be found associated with some particular form of effort, bodily or mental, or of environment. If such circumstances fail to explain the distress the probabilities are that there is some abnormal action of the heart. Inquiry then proceeds to get from the patient the first sign which called attention to there being something amiss. It may be a transient sensation such as that produced by an extra-systole, or it may be a consciousness of the heart's action called palpitation. This requires an inquiry into the mode of onset, the manner in which the heart behaved during the attack, and the mode of offset. By such inquiries one can, in the great majority of patients, recognize extra-systoles, auricular fibrillation and flutter, and paroxysmal tachycardias. The patient's account of his condition during an attack will give information as to the integrity of the heart muscle, by the presence or absence of the signs of heart failure.

The patient may have syncopal attacks. Here again the account given of the manner of onset and the patient's recovery is of great value, particularly if supplemented by a description of the patient's behaviour and appearance by an intelligent observer.

It must be kept in mind that this stage of inquiry is the most important. The facts elicited up to now give us, in many cases, the only evidences to be found of the impaired function of the heart, excepting where the impairment has gone so far as to produce dropsy and other objective signs of advanced heart failure.

I wish to insist on this point, for when the doctor recognizes the importance of this kind of information and has acquired the ability to frame the appropriate questions, and to interpret the answers, he will have obtained a knowledge of the essential matters in the vast majority of patients. Once this ability is acquired, a sound opinion can often be formed after a few minutes' time, and the subsequent examination become more or less a matter of formality.

After this the examination proceeds in the usual way, by employing inspection, palpation, and auscultation.

In using these different methods they should, as far as possible, be correlated. The pulsation in the neck should be observed at the time that the radial pulse or apex beat is being felt, and the sounds of the heart should be auscultated when the apex or the pulse is felt. In this way the timing of events becomes easy, a matter to which, unfortunately, sufficient attention is not given.

After this mechanical aids may be used, of which a description follows.

It has to be remembered that great circumspection has to be used in the interpretation of physical signs and instrumental records. Their recognition is but preliminary to the more advanced stage of assessing their value, and in doing this the facts elicited by the inquiries into the response to effort are to be correlated with them.

There are a number of manifestations, subjective and objective, at times associated with heart or other circulatory affections, such as the sense of exhaustion and syncope, which require separate consideration.

The Sense of Exhaustion and Fainting

Exhaustion in ill-health.—There are often sensations which are either unpleasant or distressful which impose a limitation on the patient's efforts. Chief among these sensations is that of exhaustion. It is a complaint made by most at the onset of an illness. It is a sensation which prevents them following their vocations with the vigour of a healthy individual.

Though little is known of the subject, that little shows that a full knowledge of exhaustion would throw a flood of light upon many obscure fields of medical knowledge, and prevent a very great deal of vain speculation and futile practice. The investigation of the subject is surrounded with difficulties, in a measure due to the fact that for its complete understanding a knowledge

of vital processes is necessary, of a kind which does not yet exist. With the imperfect knowledge which we possess some advance has been made in understanding its relation to certain diseased states, particularly in relation to cardiac inefficiency. The subject has engaged my attention for many years, and at the St. Andrews Institute a great deal of attention has also been given to it. The following brief description represents our present position.

Differentiation.—Like most other sensations, no description can convey what the sensation is like unless the reader himself has experienced it. Moreover, the reader will scarcely be inclined to accept the differentiation of this from other sensations unless he has given a good deal of attention to the analysis of his own sensations, and even then there is likely to be a lack of agreement. This difficulty is one which surrounds all inquiries into matters which require the exercise of a judgment, based on individual perceptions.

The most common sensation with which exhaustion is compared is that of fatigue, yet most people who compare the feeling of fatigue, the result of prolonged bodily effort, with the feeling which they experience during some mild febrile complaint, have little difficulty in distinguishing between the two.

A healthy man only feels fatigue after prolonged or severe exertion, but he may on occasion experience exhaustion after slight exertion, or indeed on no exertion. Fatigue is recovered from only after a period of rest; exhaustion may be recovered from, by a change of occupation or an alteration of the position of the body, or the cooling of the surface of the body. Thus, a person standing in a hot room may experience a sense of great exhaustion, but is immediately restored if exposed to a current of fresh air. A man on a sultry day wearing a heavy overcoat may feel exhausted on walking half a mile. If he strips off his coat he may be able to walk many miles in comfort, and in the end suffer, not from exhaustion, but from a feeling of healthy fatigue. A man may have a feeling of exhaustion, as, for instance, in working at some monotonous job, but he may play and enjoy a strenuous game of tennis.

Closely allied to exhaustion, and it may be produced in the same way, is the sensation of sinking and depression, which gives the impression that one is about to lose consciousness or to pass away. This depression may come on with certain other sensations, as pain due to a colic (renal, gall-stone) or angina pectoris. It also occurs with nausea, which produces vomiting, and sometimes with giddiness in certain cerebral attacks.

The mechanism of exhaustion.—This will best be understood if we study some of the simpler forms where it can be produced and recovered from. I have referred to the effect of standing in a hot room and the effect of heavy clothing in producing exhaustion. These are conditions which may produce syncope, and it is to be noted that this sense of exhaustion often precedes the syncope, and there is good reason for assuming that the cause of both is the same—exhaustion occurring at an earlier stage of the same process.

This process is simply an impaired flow of blood through the brain, the blood accumulating in the large abdominal veins or in the peripheral

vessels in the external body wall. The change from standing to the lying position or the cooling of the surface of the body at once dispels the sensation.

From such experience it would seem that the sense of exhaustion is due to a depletion of blood of the higher cerebral centres. The same conclusion is arrived at in another way. The perception of a sensation implies localization, that is, a sensation is always felt somewhere. There are certain sensations that have a very definite localization, such as pain. There are sensations so generalized that they cannot be precisely localized, such as exhaustion. That is, we recognize the sensation, but we cannot refer it to a limb or a particular region of the body, and yet we are vaguely conscious that it is present in every part of the body.

I throw out the suggestion that as in all sensations the nervous system is implicated, the more definitely localized the sensation, the nearer is the part stimulated to the peripheral distribution of a nerve. Thus the pain from a pin-prick has a limited distribution. The pain from an injury to the ganglion or the posterior root of spinal nerves has a much wider distribution, while certain visceral pains are still more widely felt, and those originating from disturbances in the brain are still more widely distributed. This is due to the fact that the higher in the nervous system a stimulus is applied, the more widely distributed will be the effect.

The function of the higher centres in the brain is impaired when the blood supply is inefficient. The result of such an impairment is bound to be widespread. The production of exhaustion can sometimes be demonstrated to be due to a deficiency in the cerebral blood supply, and the disappearance of the sensation to be due to an improvement of the blood supply.

This view is not put forward as if it had been proved, but merely as a working hypothesis, so as to give a reasonable explanation for certain phenomena and as a guide to further research.

The clinical significance of the sense of exhaustion.—When a patient states that he suffers from this sense of exhaustion we recognize that we have to deal with a condition in which there is a defective supply of blood to the central nervous system.

There are two ways in which this may be brought about—(1) by an ineffective heart-beat, or (2) from the accumulation of the blood in other parts of the body. The first of these is due to a weakened or impaired function of the left ventricle; the other is due to a disturbance of the vaso-motor mechanism.

It is necessary clearly to differentiate between these two, as the failure to recognize the distinction misleads a great many doctors, so much so that exhaustion is frequently assumed to be cardiac in origin when it is due to a vaso-motor disturbance. As a consequence the diagnosis, prognosis, and treatment are all apt to be at fault. On the other hand, the recognition that the sensation is of vaso-motor origin gives a clue to search for a cause of a particular kind.

Exhaustion due to an ineffective heart-beat.—This rarely happens, and only in extreme cases of heart failure, that is, when the reserve power of the

heart is exhausted and the heart fails to maintain an efficient circulation when the body is at rest. Thus we find it in cases of advanced heart-block, when, in addition to the block, the output of the heart is diminished. (See Observation 49, p. 272.)

It is to be noted that patients with complete heart-block, with a ventricular rate of 30, may be able for considerable effort and do not complain of exhaustion.

The sensation is also felt by patients with the small irregular pulse of auricular fibrillation. In a case of auricular flutter with a ventricular rate at times of 290 per minute, the patient's chief complaint was extreme exhaustion. The pulse was very feeble (Observation 24).

When a failing heart is able to maintain an efficient circulation when the body is at rest, exhaustion is not complained of. When a heart is so feeble that it cannot supply sufficient blood to the body when an effort is made—however slight that effort be—the sensation felt by the patient is not one of exhaustion, but breathlessness or pain.

I want to emphasize this point. It is quite possible there may be cases with heart failure, in whom effort induces exhaustion, but in my experience, when this happens there is invariably present some other factor. So that, when a patient states that effort produces exhaustion, even if there is gross disease of the heart, I take their statement as an indication that there is some agent other than the heart, or in addition to the heart, which is producing the ill-health.

Exhaustion due to vaso-motor disturbances.—The effect of posture and heat in the production of this sensation has been referred to and shown to be due to a vaso-motor effect upon the abdominal veins or the peripheral circulation.

A similar result may be produced in other ways. The heat of a febrile ailment; the toxin of a microbic infection, combined with or independent of the fever; the toxins or other agents generated from other sources, as in the exhausting diseases of other organs, are common causes.

Exhaustion and the response to effort.—In the course of an examination of a patient an inquiry should always be made as to the patient's response to effort, and particular inquiry should be made into the sensations that limit the effort. If the limiting sensation be pain or breathlessness, then the possibility of the heart being the cause has to be considered. If, on the other hand, the limiting sensation is exhaustion, then a careful scrutiny must be made for causes other than the heart—for some agent that will produce fever or toxin.

In such a search the heart may be found disturbed—usually an increase in rate. Disturbances are likely to be found in other organs, such as loss of appetite, nausea, constipation or diarrhœa, mental depression and irritability, a rise of temperature at some part of the day. Such associated symptoms indicate the presence of an agent which modifies the function of a number of organs, so that in place of attributing the ill-health to the organ whose

symptoms are most in evidence, such as the heart, we recognize we have to deal with some agent which disturbs many if not all the organs.

Fainting.—There are a number of complaints or 'attacks' which the patient experiences of a temporary kind, and more often than not the doctor does not see the patient while the attack is on, and has to rely upon the patient's description and upon replies to questions. Of these attacks faintness and syncope are among the most common. Although many of these attacks can be referred to a diminished output from the heart, the manner in which the heart fails to put out sufficient blood varies considerably.

The sudden diminution of output affects first of all the brain. A transient cessation may produce no more than a passing giddiness. In some cases there is no feeling of giddiness, but a sense of confusion and bewilderment, with a stumble if walking, or with a slurring of the speech if talking (Figs. 134 and 153). A prolongation of the cessation of output results in loss of consciousness, with a fall if standing or walking, it may be with instant recovery (Observation 59). If the output has ceased, after a few seconds convulsions appear, it may be with stertorous breathing (Fig. 183). If there is only a diminished output the loss of consciousness continues, it may be for hours, till a sufficient output is restored (Observation 32).

The effects of a diminished output or a cessation of output are not always the same. The sudden cessation of the output which results in sudden death does not cause convulsions, while the cessation which occurs in heart-block causes convulsions and sometimes stertorous breathing. In the latter case an occasional contraction may keep the brain sufficiently active to permit of the abnormal activity which results in convulsions. The effect of a cessation of output is seen in heart-block, and is described in the records of the cases of heart-block in Chapter XLII.

Other conditions produce this loss of consciousness, such as the onset of abnormal rhythm, as auricular fibrillation and flutter. (See p. 216.)

The most common forms of syncope are due to vagal excitation or the drawing of blood into the larger veins of the abdomen or of the skin.

Usually the syncopal attacks produced by the vagal action are relatively slow in their onset—the patient is conscious of the oncoming and seeks support, or the sight is first affected and 'all becomes dark'. The heart beats slowly and weakly and convulsions rarely appear, though Laslett has recorded a case with tracing where standstill occurred and convulsions followed.

As a rule syncope of vagal origin is due to some cause, mental or physical —the hearing of alarming news; one patient lost consciousness and became convulsed when I told him he had typhoid fever. That was twenty years ago, and he is alive and well to-day. Others faint at the sight of blood, or on some trivial operation as opening an abscess. The vagal origin of the syncope can often be inferred from some other phenomena in which the vagus is involved, as vomiting, diarrhœa, or the occurrence of a large loose motion some hours later. In this last instance it is assumed that there had been some commotion in the bowel which caused the syncope. Attacks of colic,

as renal colic, may be accompanied by great weakening of the pulse and slowing due to vagal stimulation and accompanied by vaso-motor disturbance —pallor and sweating.

Syncopal attacks are often due to draining of the blood in the large abdominal and peripheral veins. This probably accounts for the fainting of people when standing in a warm and still atmosphere, as in crowded rooms and churches. Some people have a great tendency to this form of fainting. In many the presence of this stasis in the abdominal veins can be demonstrated. In most people pressure by the palm of the hand on the abdomen when lying down causes little or no distension of the veins of the neck. On others the veins of the neck swell visibly; sometimes they become very large (Fig. 281). One patient consulted me because he found that after sitting for some time, he sometimes fainted on resuming the erect position. On examining him I found that pressure on the abdomen produced an enormous distension of the jugular

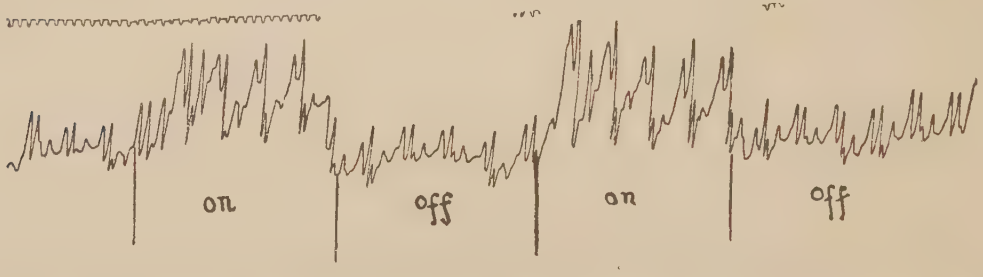


Fig. 281. Tracing of the jugular pulse while pressure was applied to the abdomen (*on*). The jugular vein increased in size, while on removal of pressure (*off*) the vein became less distended.

veins. I told him to wear an abdominal belt firmly applied, and explained the reason for this, and he said that he had found that before standing up, if he bent forward, held his breath, and compressed his abdomen, he did not faint when he stood up. I have seen a goodly number of people in whom the tendency to syncope disappeared when they wore a suitably applied abdominal belt. Thus a tall lady was liable in hot summer weather to syncopal attacks. When cycling she had sometimes to get off her bicycle and lie down to avoid an attack. An abdominal belt gave her complete relief, and she found the attacks tended to recur if she left off wearing it.

I do not understand the whole of the mechanism which brings about syncope in these cases. In a patient in my clinic Dr. Marris made a number of interesting observations. This man felt quite well when lying down, and nothing wrong could be detected on physical examination. But as soon as he stood up he became giddy and faint, and had to lie down. His heart-rate when lying down was about 60 per minute, the pulse of good strength. When he stood up the rate increased to 120, and became very small and weak. An abdominal belt gave him such relief that he was able to follow his occupation.

The explanation of the phenomena of exhaustion and syncope involves a knowledge of the structures and processes concerned in regulation of the blood-vessels—arteries, capillaries, and veins, and that knowledge is far from being sufficient for the purpose.

The point to bear in mind is that these are rarely signs of a heart affection.

CHAPTER LXIV

Instrumental Methods of Examination

The sphygmograph.
The polygraph.
The clinical polygraph.
The ink polygraph.
The electro-cardiograph.
The electro-cardiogram.

In the examination of the vast majority of patients the diagnosis can be made independently of instrumental methods. It must not be inferred from this that graphic records can be dispensed with, for the power to diagnose the great majority of cases comes through the information obtained by this means. Though it is not necessary for a physician himself to take records, he must be familiar with their interpretation in order that he may appreciate and apply the results.

There have been many methods devised to record the movements of the circulation, but here I will only deal with those which in my hands have yielded satisfactory results. The essence of a method should be simplicity, for the more complicated the processes the more unsuitable it becomes for practical clinical purposes. In hospitals with large staffs of assistants, the more elaborate methods may be usefully employed, but for the practitioner who studies his own patients, the simpler the method the better.

The sphygmograph.—It is scarcely necessary to enter into a full account of the construction of the various sphygmographs. They have been so frequently described in text-books that their construction is familiar to all medical men. They are all practically constructed on the same principle. A steel spring is laid upon the radial artery at the wrist in such a manner that, while it compresses the artery, it does not obliterate it. Attached directly to the spring is a long lever, or a series of small levers, that magnifies the movements of the spring. The free extremity of the lever presses lightly against a strip of paper, whose surface has been blackened by the smoke of burning camphor or turpentine, the strip of paper passing at a uniform speed by means of a clockwork arrangement. Although I have worked with several instruments, I find the Dudgeon to be the handiest and most useful. Into all sphygmographic records, certain errors, due to defects of the instruments, creep. Some of the more elaborate instruments may be freer from defect than the Dudgeon; but so long as one is cautious not to read

into the tracings movements evidently due to instrumental errors, the Dudgeon sphygmograph is quite serviceable for a great many practical purposes, and more particularly for giving a true and accurate record of the occurrence of pulse-beats.

The polygraph. — There are many perceptible movements due to the circulation that the sphygmograph fails to register; and when it is required to record these movements other instruments have to be employed. The method most commonly adopted has been by conveying, by means of a tube containing air, the movements to be registered to a tambour on which there rests a lever. The excursion of the lever is recorded on a revolving drum covered by smoked paper. Two or more tambours being used with their levers placed one above the other, the simultaneous record of different movements can be readily effected.

The elaborate and bulky apparatus required has restricted the employment of this method to such narrow limits that numerous points of interest in clinical medicine have been either overlooked or misunderstood. In my investigations into the nature of the venous pulse, I had at first to use this unwieldy instrument; but its cumbersomeness compelled me to devise a much simpler and more effective apparatus.

The clinical polygraph.—I attached a tambour to a Dudgeon sphygmograph in such a manner that it was possible to obtain at the same time as the radial pulse was being recorded, some other movement as the jugular pulse, the carotid pulse, the apex beat, and the pulsation of an aneurysm. This instrument was extremely convenient, and with practice I became so adept that records could be taken in a few minutes. The instrument could be carried in the pocket, and when visiting patients in their homes records could be obtained while the temperature was being taken. In Fig. 282 there is shown the Dudgeon sphygmograph with the tambour attached. The band supplied to bind the instrument to the wrist is rigid, with a rather inconvenient form of buckle. It was found more convenient to replace this rigid band by an elastic band and tie the ends in a simple knot. By this means the position of the instrument can be shifted to obtain a good record. Fig. 283 is a drawing of the tambour with the lever of thin aluminium or other light material. The stem by which it is attached to the sphygmograph is shown. The extremity *C* is bent at right angles to fit into a slot on the upright stem of the sphygmograph. To adjust the lever to the required position, three movements are obtained by the sliding arrangement at *D*, and by the joints at *E* and *A*. By means of these arrangements the lever can be moved with great facility in a vertical or horizontal direction as well as made to approach or recede from any given point. By this method can be recorded various movements at the same time and on the same paper as the radial pulse. The lever can be made to write directly above or below the writing lever of the sphygmograph, or when the latter movements are large, a little behind, so that the two levers do not come into contact during their excursions. Perpendicular lines by the lever should be made at the beginning or end of

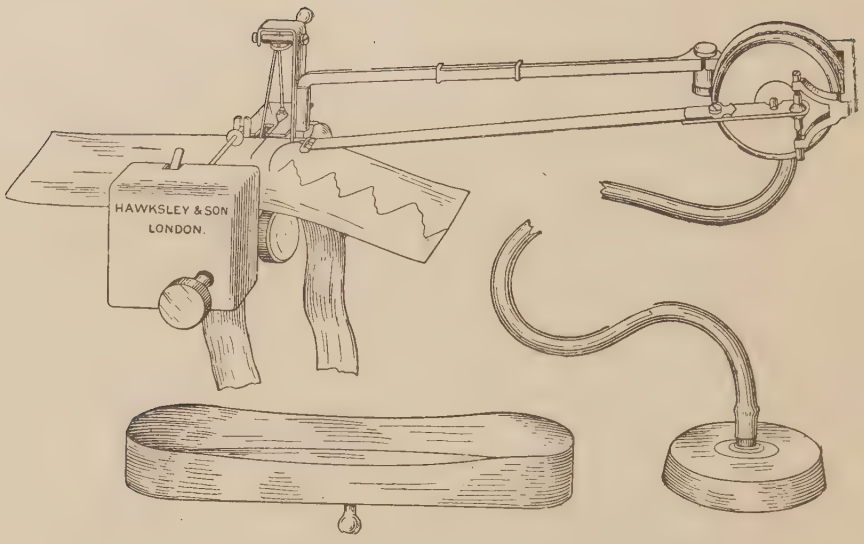


FIG. 282. The clinical polygraph, consisting of a tambour attached to a Dudgeon sphygmograph.

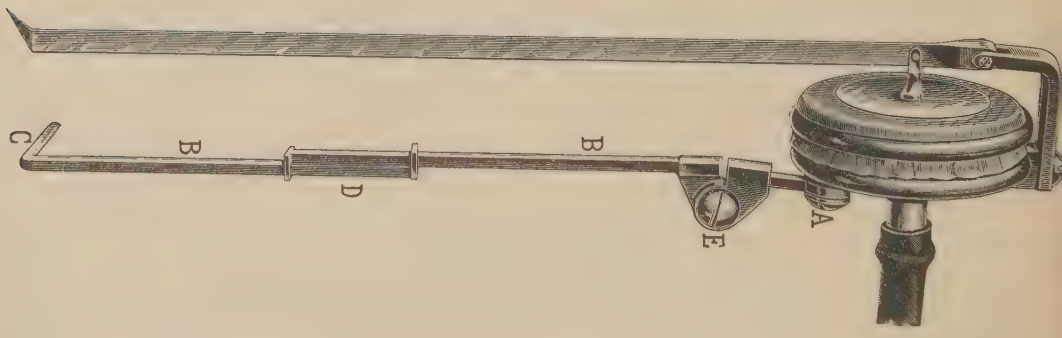


FIG. 283. The tambour with stem for attachment to the Dudgeon or Jacquet sphygmograph.

the tracings, so that points are obtained from which by a pair of compasses the relative time of any event can be accurately gauged (see Figs. 284, 285).

I retain a regard for this simple device, for it was by it I was enabled first to obtain an insight into the heart's movements. It records essential matters such as the character of the radial and jugular pulses better than any other instrument. The great store of records that I have collected have furnished me with the facts upon which I have been able to perceive the functions of the genetic system, affording opportunities which could be found nowhere else.

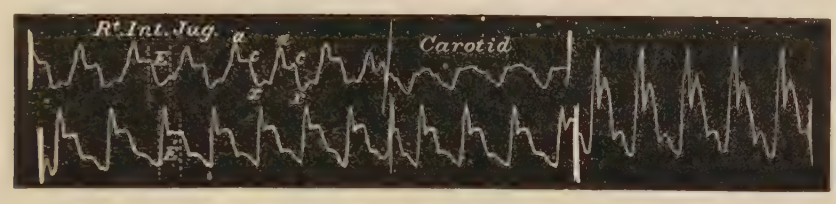


FIG. 284. The first part of the tracing shows the venous pulse from the right internal jugular vein (upper tracing) taken at the same time as the radial pulse (lower tracing). The clockwork being stopped, the receiver was applied over the carotid artery, and tracings of the carotid and radial pulses taken. Again the clockwork was stopped, and the tambour portion of the polygraph removed, and the full effect of the radial pulse was inscribed on the last portion.

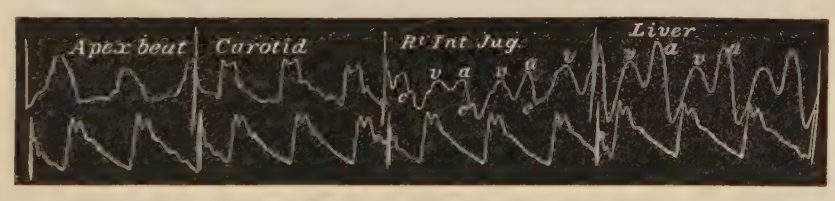


FIG. 285. Apex beat, carotid, right internal jugular, and liver pulses (upper tracing) taken at the same time as the radial pulse (lower tracing).

The ink polygraph. — Simple and useful as the clinical polygraph was found, it was not convenient when movements had to be recorded over long periods, as when irregularities were infrequent or varied, or when the effect of a drug or effort, or respiratory movements had to be noted. I therefore conceived the idea of constructing an instrument that would take tracings for long periods. This idea resulted in devising an instrument where a form of pen with ink could be employed to write on a roll of paper as it was being unwound. This instrument is shown in Fig. 286.

The essential parts of the instrument are a small cup (*E*, Fig. 286) for receiving the impressions of the pulsations, a tube for transmitting the impressions to a tambour (*B*, Fig. 286) and lever, with pen (*F*, Fig. 286).

The small cup for receiving the impressions (which will be referred to hereafter as the ' receiver ') is simply a small shallow vessel, circular in shape, one and a half inches in diameter and half an inch in depth. The open

mouth is applied over the pulsating part so that its edges are closely adapted to the skin ; and all communication with the outer air is excluded. From the roof of the receiver rises a narrow pipe, half an inch in length. To this

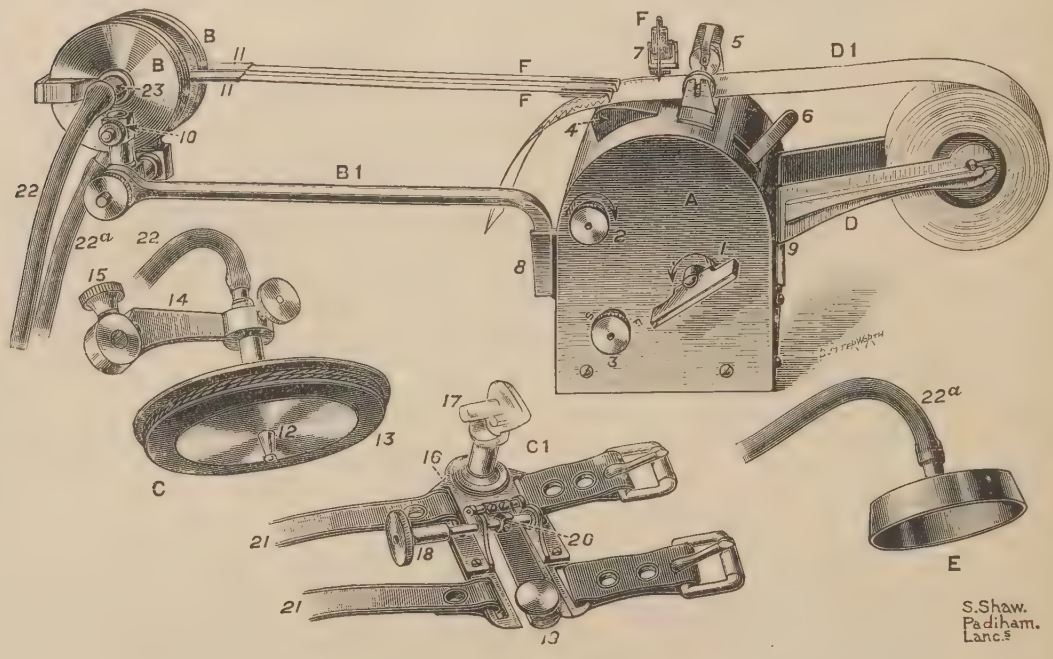


Fig. 286. The ink polygraph.

pipe is fitted an india-rubber tube three to four feet in length, the other end of which is connected with the tambour. A modification of this receiver is required when tracings of the liver pulse are taken. The ' liver receiver ' (Fig. 287) is larger, being five inches in length, two inches in breadth, and

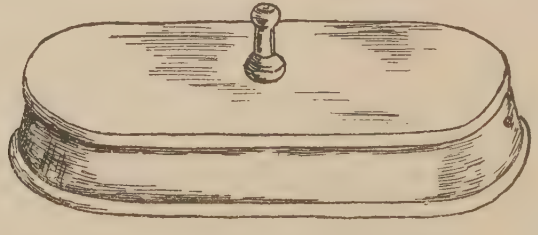


Fig. 287. The liver receiver (half size).

one inch in depth, its open edges slightly curved on their long axis. A small air-hole is made at one end near the roof. In employing the ' liver receiver ', the position of the lower margin of the liver having been ascertained, the receiver, held in the right hand, is laid lengthwise across the abdomen, its

lower edge being two inches below the liver margin, and the end with the air-hole towards the middle line. Steady continuous pressure is applied to the lower margin of the receiver till it presses deeply into the abdomen; and then the upper margin is adapted closely to the skin. In this manner, a considerable portion of the lower liver edge is embraced by the receiver. If the forefinger of the right hand is now applied over the air-hole, the movements of respiration and liver pulse will be communicated to the lever. If the patient stops breathing, the liver movements are alone transmitted.

The case *A* (Fig. 286) contains the clockwork for the roller which unwinds the roll of paper *D*, and also the separate clockwork which moves the time-marking pen *F*. *B B* are the two tambours, and *F F* their levers. The writing pens in Fig. 286 are flat aluminium levers with a writing point at one end at right angles to the lever. The ink is placed, by means of a small camel-hair brush, in a shallow groove immediately behind the writing point. The writing point is adjusted so as barely to touch the paper. If the pens are kept clean, and the ink is free from dust, they serve their purpose most admirably, and are ever ready for use. Red ink is better than the black inks, as it does not corrode the pens. As the radial pulse is the most serviceable of standards, a special method is employed to record it. A splint (*C* 1) is fastened to the wrist in such a manner that the pad of the steel spring falls on the radial artery and is pressed down by an eccentric wheel (18), until a suitable movement is transmitted to the spring by the artery; then the broad tambour (*C*) is fitted on to the splint so that the knob (12) falls on the moving spring. This wrist tambour is connected to the tambour *B* by india-rubber tubing (22, 22*a*), and the movements of the radial pulse are recorded by the lever *F*. The shallow cup (receiver) *E* is placed on the pulsation which it is desired to record; and the movement is conveyed to the lever *F* of the other tambour. In this way, simultaneous with the radial pulse, a record can be obtained of the apex beat, carotid, jugular, or other pulses.

To record the respiratory movements, a bag can be substituted for the receiver *E*.

By turning the screw (3) the rate at which the paper passes can be quickened or slowed at will. This is of the greatest use; for it often happens that in quickly succeeding events a wider interval may be required, whereas in recording respiratory movements a slow rate is best. As the time-marker registers one-fifth of a second, and is driven by a separate clockwork, the rate of the recorded movements can always be ascertained with absolute accuracy.

It has been suggested that another tambour should be added to record a third movement; and I have tried this, but have practically discarded it, as, though it might be of use occasionally, it would complicate the apparatus unnecessarily. When one is making observations single-handed, the two tambours are quite sufficient to occupy the attention. With a little practice, this apparatus can be used with the greatest facility. In the course of a few

minutes the different movements can be recorded, with the patient sitting up, or in the recumbent position.

When the tambour is strapped to the wrist to take the radial pulse, one hand is always free to start the machine, and to replenish the ink or regulate the rate, the other hand holding the receiver over the movement to be recorded.

The electro-cardiograph.—It has long been known that the contraction of muscle gives rise to electrical changes. Waller pointed out that if the legs or arms were used as leads and connected with a galvanometer, the electrical reactions which occur with the contraction of the heart would affect the magnet.

Einthoven invented the string galvanometer, by means of which the movements of the heart can readily be registered. In this instrument a thread of platinum or other suitable material is placed between the poles of a magnet. As it lies in this magnetic field, the thread becomes very sensitive to the slightest electric change. The right arm and left leg or right arm and left arm are immersed in a suitable basin containing salt and water, in which there is a sheet of copper. This sheet of copper is connected by wires with the thread lying between the poles of the magnet. The electric disturbances caused by the movements of the heart chambers are conveyed to the thread and cause it to execute a very slight movement. A series of lenses is so arranged that, by means of an arc light, the shadow of the thread is thrown upon a movable photographic plate, in such a manner that the movement of the thread, when influenced by the electrical changes of the heart, is photographed.

The electro-cardiogram obtained from a normally acting heart shows a series of movements due to the contraction of the auricles and ventricles (Figs. 288 and 289). In a cardiac revolution, the first wave *P* is due to the contraction of the auricle ; this is followed by a series of waves, the result of the ventricular contraction. The first of this series is a slight depression (Fig. 291), followed by a steep sharp rise *R*, which falls equally rapidly, and is followed by a negative wave *S*. This is again followed by a slower rising wave *T*, of less height than *R*.

While the waves shown in Fig. 288 are present in all normally acting hearts, they may show slight variations.

The electro-cardiogram.—In the description just given it is stated that the waves P and R in the electro-cardiogram are due to the contraction of the auricle and ventricle. According to the description given by authorities, this is not true, as it is accepted that the electro-cardiogram is a record of an ' excitation wave ' which is described as ' a wave of altered electrical conditions which flows over muscle *preparatory* to its contraction '.

When it is inquired whence comes this force or wave of excitation we find that no answer can be given. The consequence is that there is a great deal of confusion in the interpretation of the electro-cardiogram and in the conception of the heart-beat. In recent years the instrument is being used

more and more in the investigation of a great many conditions, both clinical and experimental. When the outcome of this work is scrutinized there are revealed a few facts of value and a great deal which is little better than conjecture. As it is recognized that somehow or other the movements recorded

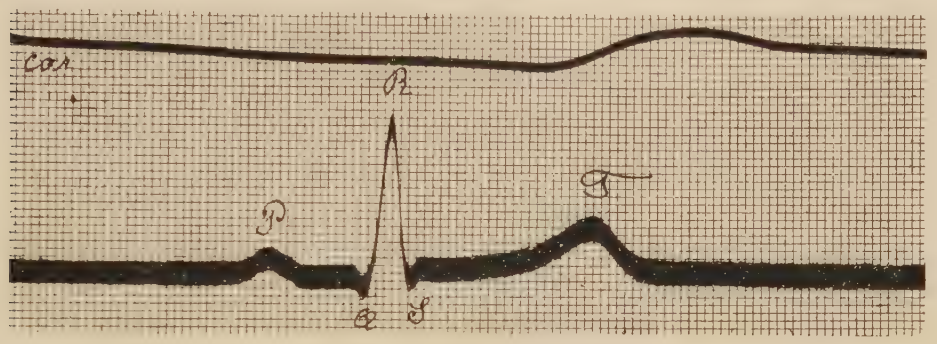


FIG. 288. Shows a portion of a carotid curve and a single beat of an electric curve. The abscissæ are divided at intervals of 0·01 sec.; the ordinates are divided at intervals of 10^{-4} volt. The lead was from right arm and left leg. In this, corresponding points of time are directly vertical to each other. *P* represents the auricular and *Q*, *R*, *S*, and *T* the ventricular contraction. (Einthoven.)

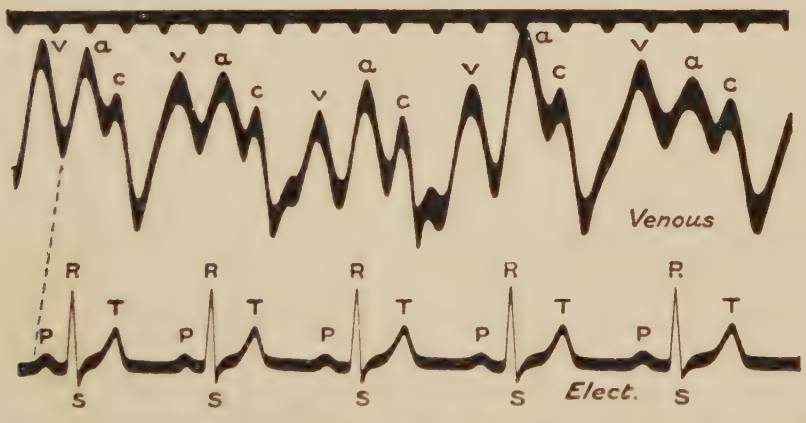


FIG. 289. Simultaneous records of time, in one-fifth second, venous and electro-cardiographic curves. The delay in the venous curve is due to the air transmission employed, to the delay in transmission from auricle to neck, and to the fact that the electric change slightly precedes the contraction. From a patient in whom the *P-R* and *a-c* intervals show slight prolongation, but in whom the curve is otherwise normal.

are associated with the movements of the heart, the relative times of occurrence of auricular and ventricular systole can be determined. It is found also that the shape of the waves is fairly constant in the normal heart, and that certain variations can be recognized as peculiar to certain abnormal contractions of auricle and ventricle. From such knowledge some light can, in rare instances,

be thrown upon abnormal actions of the human heart, but beyond this the great majority of the variations are beyond interpretation, and have become a field for much vain speculation.

One of the consequences is that an instrument is used which records events whose origin is unknown. These records of unknown events are used to interpret the contraction of the ventricle. On the other hand, the knowledge of the contraction of the ventricle is very imperfect, and this very imperfect knowledge is used in its turn to interpret the electro-cardiogram. The employment of an instrument for a given purpose requires that it should be known what it is that the instrument reveals. If this is not known the conclusions drawn are certain to be misleading.

Being conscious of my ignorance of physics, I have hitherto refrained from expressing any views about the electro-cardiogram. In the following remarks I describe what seems to me a common-sense way of looking at the matter. I do not, for a moment, pretend that this description will be in all respects correct, but I would rather make an attempt and fail than merely use a jargon which is meaningless to me and to my readers. Moreover, this attempt at an explanation is a challenge to those with a fuller knowledge of physics to supply a better interpretation.

The view that I take is, there must be a cause of the ' excitation wave '.

The functioning of a cell is the result of chemical changes. When a muscle cell functions two effects are capable of demonstration. One is the discharge of a force or a form of energy which is capable of producing electrical changes which can be made to move the string of a galvanometer. Such movement is that recorded by the electro-cardiograph.

The other demonstrable effect is the contraction of the muscle cell. The *contraction* does not produce the force any more than it produces the ' cell impulse '.

This description implies that the force producing the electro-cardiogram and the contraction are the results of energy discharged by the muscle cells. The objection to this explanation is that in timing the manifestations of the electro-cardiogram and of the contraction of the ventricle, that the former precedes the latter by a minute space of time, and consequently they could not both arise from the same cause. The assumption that these two results could not have the same origin is based upon a fallacy. The timing of events in nature which are different in character cannot be compared unless all the factors concerned in the production of each event are known. When a gun is discharged an observer at a distance sees a flash of light before he hears the sound. If he took the sound as a record of the time of explosion then the flash of light would be supposed not to be due to the explosion, but would be a ' wave preparatory to the explosion '.

The proof put forward that the force producing the electro-cardiogram precedes the contraction of the muscle, is based upon a simultaneous record of this force and the effects of the contracting muscle. The movement recorded in the electro-cardiogram is due to a force instantaneous in its dis-

persion. In this regard it is evidently of the same nature as the force which produces sound which is instantaneous in its dispersion, while the sound waves it sets up take time in their dispersion. A man speaking at one end of a hall 300 feet in length can be heard 300 miles away by means of a wireless telephone sooner than he can be heard by an individual at the other end of the hall. The reason is that the force is instantaneous in its dispersion, so that a man 300 miles away with a wireless telephone at his ear hears the sound because the sound waves have only to travel a few inches, whereas the sound waves from the man's voice in the hall have to travel 300 feet to the listener at the end of the hall.

The other effect of the muscle cell function is contraction. This is a vital process which takes time in its performance, consequently any manifestation of contraction is bound to be later than a manifestation produced by a force which is instantaneous in its dispersion.

Of these manifestations of the contracting muscle the sounds of the heart are taken to indicate the beginning of the contraction of the ventricle, and electrical records of these sounds have been shown to appear at a minute period of time later than the record of the movements of the electro-cardiogram. If the factors concerned in the production of the sounds be considered it will be found that such a difference in the time relations is bound to occur. The beginning of a ventricular systole is due to a few cells contracting. The number of cells contracting rapidly increases, producing the phenomenon which is recognized as the beginning of ventricular systole. With this contraction pressure within the ventricle begins to rise. This causes the valves to close with such tension that sound waves are produced. These waves travel a short distance through solid tissues and the air, and raise a membrane which sets up an electric current. All these events take time, and are quite sufficient to account for the difference between the time of the two records. The conclusion then is that the two events are synchronous in origin, but the manifestation of the one precedes that of the other.

Another error has arisen from seeking to interpret the electro-cardiogram by means of an imperfect knowledge of the contraction of the ventricle. A strip of muscle fibres when electrically stimulated to contract gives rise to certain electrical changes which can be recorded. The records of these changes are taken as the basis for interpreting the electro-cardiogram under the impression that the muscle fibres of the ventricle contract in the same way as the strip of muscle. It will be observed in this experiment that all the fibres are stimulated to contract simultaneously, and when they have contracted, no more cells are active, and the contraction is momentary, like a twitch. Electrical changes occur at the beginning of the contraction and at its cessation, but in the normal contraction of the ventricle the muscle cells do not suddenly contract and suddenly cease contraction all at once, but groups of cells contract in relays, and the contraction is maintained for a definite period. When any part of the ventricle passes into contraction in this way it remains contracted during the whole period of systole, and the discharge of energy from each

portion of the heart is continuous, and therefore the electro-cardiogram is not an expression of individual parts of the ventricle such as the apex or the base, but represents a discharge of energy in which every part of the ventricle participates. Therefore it is not correct to speak of an ' excitation ' wave, or a ' contraction ' wave passing in this direction or that.

Imperfect as this description may be found, nevertheless it is based upon a certain realization of the principles which govern the activity of organs, which has been described as the ' law of fluctuation '.

Our observations so far clearly indicate that until this law is employed in the interpretation of such phenomena as muscle contraction, the whole matter of the heart-beat and the explanation of the electro-cardiogram will remain little more than guess-work. Already the history of the electro-cardiogram, brief as it is, shows how vain and futile have been many of the interpretations of the records taken from the human heart.

CHAPTER LXV

The Position and Movements of the Heart

The position of the heart.
The standards for recognizing the events in a cardiac revolution.
Conditions of the chest-wall permitting the recognition of certain movements of the heart.
The nature of the movements graphically recorded.
The apex beat.
Interpretation of a tracing of an apex beat due to the systole of the left ventricle.
The auricular wave.
Retraction of yielding structures in the neighbourhood of the heart during ventricular systole.
Liver movement due to cardiac aspiration.
Epigastric pulsation.
The apex beat due to the right ventricle.
Significance of the inverted cardiogram.
Alteration of the apex beat due to retraction of the lung.
The shock due to the ventricular systole.

The position of the heart. — Professor Waterston has lately been re-investigating the position of the organs in the human body; and he has furnished me with the following result of his observations on the heart:

'Both the position and the form of the heart present considerable variation in different individuals of the same age and sex, hence it is impossible to lay down a rigid statement of what is normal in the position or form, except within fairly wide limits.

'It may be stated, first of all, that in the female the heart is slightly smaller than it is in the male, and that in old age there is a diminution of the size of the heart which may be considered to be normal. In the child the heart lies at a somewhat higher level than in the adult—this difference being possibly associated with the large size of the liver in the child.

'Apart from these variations associated with age and sex, the individual variations are probably to be associated with the different shapes of the chest, and with the varying height of the diaphragm, which are found in different individuals.

'I have recently made a number of observations upon the position of the heart as a whole, and of its chambers in relation to the chest-wall, by a method of projecting orthogonally the outline of the ribs, the sternum, and heart on to a flat surface.

'Fig. 290 shows an average result obtained in this way, from an adult male. It will be seen, from the figure, that the anterior surface of the

heart is of a quadrilateral shape. The whole of the right border is formed by the right auricle, and the same chamber constitutes the whole of the heart which lies on the right side of the sternum. The right ventricle con-

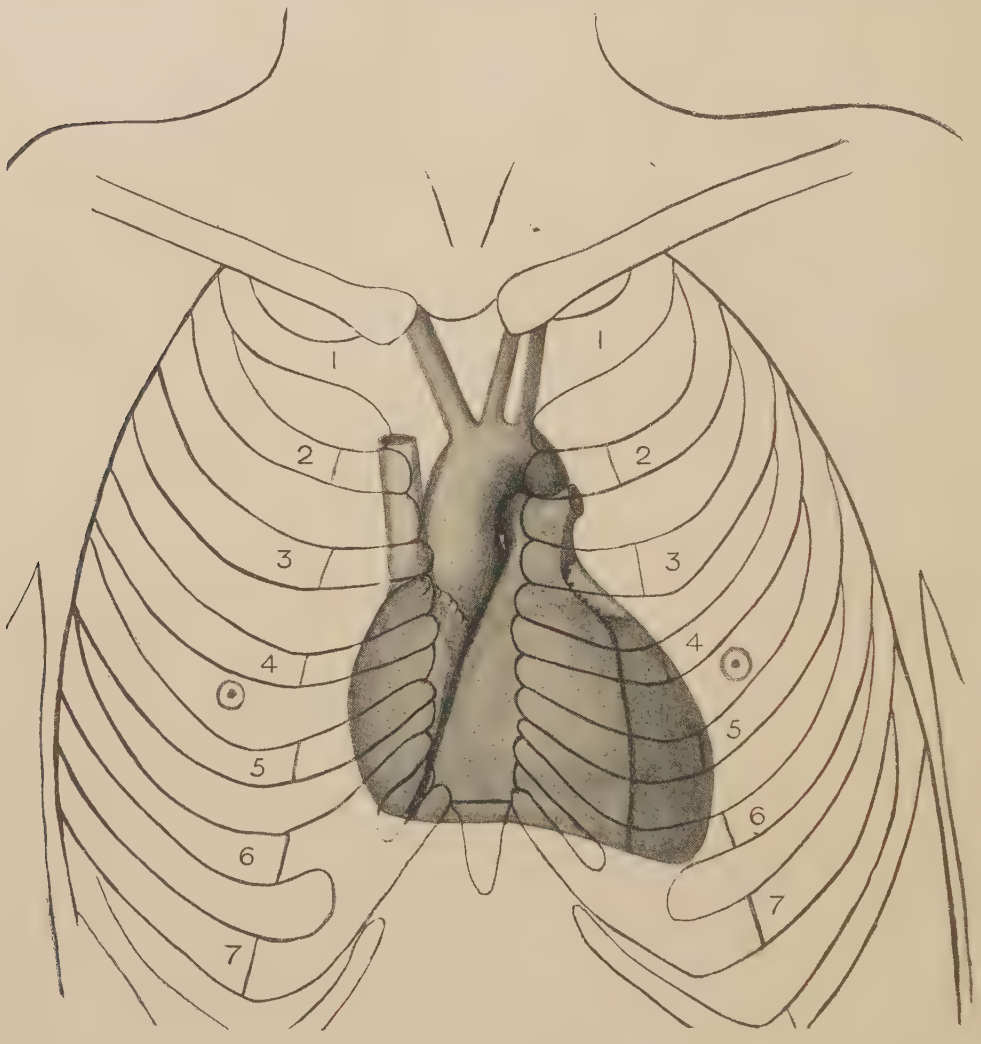


Fig. 290. An accurate delineation of the relation of the heart to the anterior chest-wall obtained by orthogonal projection. (Waterston.)

stitutes the greatest part of the anterior surface and lies behind the sternum and to its left, while the left margin and the apex of the heart are formed by the left ventricle. Only a very small portion of the left auricular appendix appears on the anterior surface, at the root of the pulmonary artery, and it is quite insignificant.

'It is quite usual to find the apex of the heart behind the sixth costal cartilage, and the lower margin of the heart is frequently found at a lower level than is usually given, namely the lower border of the body of the sternum ; and it descends behind the ensiform cartilage.

'The greatest vertical and transverse measurements of the projected outline of the heart measure four and a half to five inches in the adult male. Frequently, however, one of these dimensions is increased, usually the vertical, while the transverse diameter is somewhat diminished. This shape of the heart is associated usually with a long narrow chest.

'These observations have been made on the cadaver in the horizontal attitude, and may be taken to indicate accurately the position and shape of the heart in that condition.

'If these outlines are compared with the outlines taken during life in the vertical position, by the orthodiagraph (Fig. 291), it is found that there is a difference in the results obtained, indicating that the position of the heart is different. In the vertical attitude, during life, the heart lies at a lower level, and the lower border of the heart descends behind the ensiform cartilage to a varying degree, and the right ventricle comes to lie behind the upper part of the abdominal wall in the infracostal angle. This descent is associated with the descent of the diaphragm which occurs in the change from the horizontal to the vertical position.

'It is further found that the movements of respiration affect the position of the heart very considerably, and especially the level of the lower border, which rises in expiration and sinks in inspiration. The upper border, on the other hand, is not so much altered, but the right and especially the left borders are influenced especially at their lower ends. The left margin, as is shown by Fig. 291, was found in expiration to move to the left side to a distance of as much as one inch.'

The standards for recognizing the events in a cardiac revolution.—Owing to their easy recognition and determined place in the cardiac cycle, the carotid and radial pulses form the most certain standards for finding out the place of other movements in a cardiac revolution. In describing the tracings frequent references will have to be made to these standards, and more particularly to that period during which the semilunar valves are open, which is indicated in some tracings by the space *E*. When it appears in the radial tracing, it corresponds to the effects of the ventricular systole upon the radial pulse—that is to say, to the actual pulse-wave—and not to the true time of the occurrence of the ventricular systole, for the pulse-wave having a longer distance to travel, the period *E* will be later in the radial than in the apex or carotid tracings.

Conditions of the chest-wall permitting the recognition of certain movements of the heart.—The movements of the heart in a healthy person are often so obscured by the lungs, that only very little change is discernible in the external chest-wall. In many cases the lungs are so voluminous, or the chest-wall so fat and thick, that no movement can be detected.

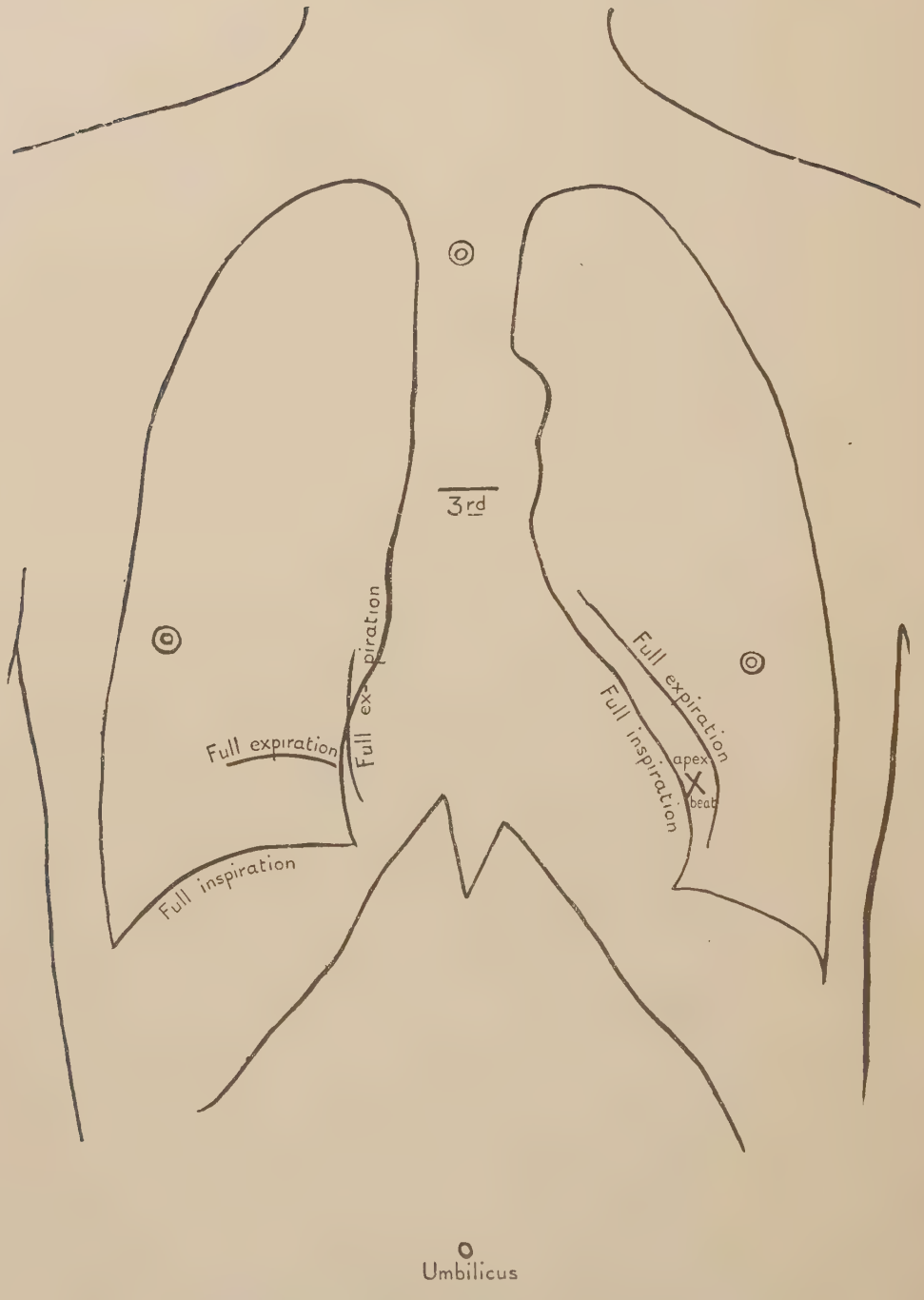


Fig. 291. The position of the heart and blood-vessels in the chest obtained by the orthodiagraph. (Ritchie.)

But when a large surface of the heart is directly exposed to a thin chest-wall, the heart being normal or increased in size, and the lung displaced, a series of movements of the chest-wall due to the contraction and expansion of the heart can be recognized. The movements thus discernible are not the same in all cases, but depend on which part of the heart's surface comes in contact with the chest-wall and other yielding structures. These movements take place so rapidly that it is difficult to interpret their significance by the unaided senses. Many writers on this subject have drawn elaborate, but nevertheless erroneous, conclusions from such unaided observations; and it seems to me that accurate observations by the graphic method alone can furnish a clear and definite explanation.

The nature of the movements graphically recorded.—The movements of the heart that are most readily recognized are those connected with the systole and diastole of the ventricles. Movements directly due to the auricles are so obscured by the larger and more vigorous movements of the ventricle that it is doubtful if they are ever capable of recognition. The movements most readily recognized are: first, the apex beat; second, the filling of the ventricles; third, the emptying of the ventricles; and fourth, the shock communicated by the sudden hardening of the ventricular walls as they pass into systole.

The apex beat.—The chief movement, and the one which is usually most apparent, is that caused by the forcible outward projection of the apex of the heart during the ventricular systole—the apex beat. This is generally described as 'the lowest and outermost point of the heart which strikes against the chest-wall'. In healthy adults it is usually felt in the fifth left intercostal space, immediately inside the nipple line. It may, however, be situated in the fourth interspace, and outside the nipple line in children and in some adults. In disease of the heart it alters its situation with the increasing size of the heart. This forward thrust occurs when the left ventricle is in contact with the chest. As will be shown later, a movement of another description occurs when the right ventricle constitutes the so-called apex beat. That the apex beat due to the left ventricle is a distinct displacement of the heart forward, can be recognized by the senses of touch and sight.

It should be kept in mind that the 'apex beat' which is found in the clinical examination is not the real apex beat. In Fig. 20 the real apex is behind the 6th rib, while the beat can only be felt in the fifth interspace. Moreover, the real apex is often hidden behind the enveloping lung, particularly when there is much enlargement of the right heart—the left ventricle being pushed so far back that all that portion of the heart which approaches the chest-wall is the right heart—the left ventricle forming only a strip, covered over by the lung. The apex beat is then due to the right ventricle as in Fig. 295 (see Figs. 247 and 248).

During the whole time occupied by the systole of the ventricle, the apex is usually kept projecting into the interspace, so that the palpating hand

recognizes the forward thrust, and in such a tracing as Fig. 292, the lever taking the tracing is kept raised during the whole time of the outflow from the ventricle (space *E'*). If the left ventricle is much hypertrophied, the same movement can sometimes be detected in two or three interspaces. If the interspaces be fairly open and the chest-wall thin, and the tip of the finger be thrust into the third or fourth interspace near the sternum, the right ventricle can be felt hardening, and remaining thus hardened in contact with the finger during the whole period of the ventricular systole. It cannot, however, be averred that in this case there is a forward thrust. The heart here is always in contact with the chest-wall, and the finger pushed into the interspace during the diastole impinges, in all likelihood, against the lax ventricular wall. As soon as the ventricle hardens, the finger recognizes this hardening as something pushing against it. This sensation of a thrust is sometimes actually synchronous with an indrawing of the soft structures filling up the interspace (Fig. 298).

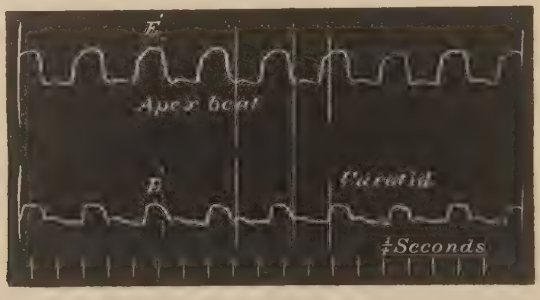


Fig. 292. Simultaneous tracings of the apex beat and the carotid pulse, showing the 'systolic plateau' in the cardiogram during the outflow from the ventricle (*E'*).

Interpretation of a tracing of an apex beat due to the systole of the left ventricle.—A tracing of the apex beat or cardiogram is a diagrammatic representation of (*a*) the forward movement of the apex of the heart, while the ventricular muscle is beginning to contract (space *D*, Fig. 293); (*b*) the retention of the apex beat against the chest-wall while the ventricles are emptying (space *E*, Fig. 293); (*c*) the backward movement of the apex of the heart, while the ventricular muscle is relaxing (space *F*, Fig. 293); (*d*) and the gradual filling of the ventricle during diastole (space *G*, Fig. 293).

(*a*) *The period of commencing contraction of the ventricular muscle—the presphygmic interval* (space *D*, Fig. 293).—During this period the pressure within the ventricle is rapidly rising. The auriculo-ventricular valves close as soon as the pressure within the ventricle rises above that in the auricle, and the semilunar valves open as soon as the pressure in the ventricle rises above that in the aorta. This last occurs at the end of the period *D*, Fig. 293, and is usually indicated by the abrupt termination of the upstroke.

In Fig. 293 simultaneous tracings were taken of the pulsation in the

pulmonary artery and of the apex beat. As the beginning of the pulse in the pulmonary artery indicates the opening of the semilunar valves, so it is found that the end of the period *D* corresponds exactly with the beginning of the pulsation of the pulmonary artery. When this figure was taken, the cylinder was rotated rapidly, in order to separate the events as widely as

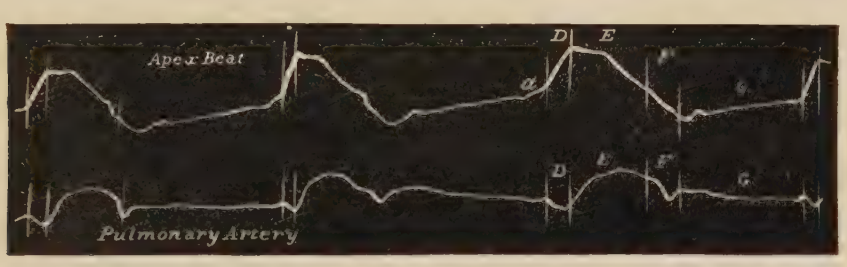


FIG. 293. Simultaneous tracings of the apex beat and of the pulsation in the pulmonary artery. *a* represents the small wave due to the auricular systole. The time during which the ventricle is passing into systole is represented by the space (*D*), emptying (*E*), relaxing (*F*), filling (*G*). While this tracing was being taken the cylinder was rapidly rotated. The letters *D*, *E*, *F* have reference to the same periods in the cardiac revolution as in Fig. 317.

possible. When the cylinder rotates at a slower rate, this period is represented by an almost perpendicular line (Fig. 294).

It will be noted that the termination of the upstroke corresponds exactly with the beginning of the outflow of the ventricle into the artery. These tracings were taken from a lad, suffering from phthisis of the left lung, which had retracted from the heart, and through the thin chest-walls the various movements could easily be observed. In the second left interspace there was a marked pulsation, and tracings of this, taken at the same time as the

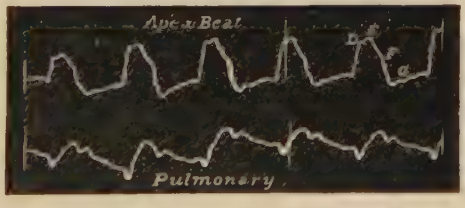


FIG. 294. Simultaneous tracings of the apex beat and of the pulsation in the pulmonary artery. The letters have the same significance as those in Fig. 293.

carotid pulse, left no doubt as to its being caused by the pulmonary artery (Fig. 295). It will be noticed that the carotid pulse appears just a very little later than the pulmonary. Here also, after a few beats, the cylinder was rapidly rotated, with the hand, to separate more widely the different events.

(*b*) *The period of ventricular outflow* (space *E*, Fig. 293).—When the pressure in the ventricles exceeds that in the aorta and pulmonary artery the semilunar valves open, and the blood flows out from the ventricles.

During this period the apex is usually kept stationary, pressing against the chest-wall, and in many tracings (as in Fig. 296) it is shown by a fairly level line—the 'systolic plateau'. In place of a flattened top representing the period of ventricular outflow, the tracing sometimes rapidly descends (Figs. 293 and 294). I cannot but think that this is due to the ventricle shrinking away from the interspace during its systole, the receiver perhaps not being

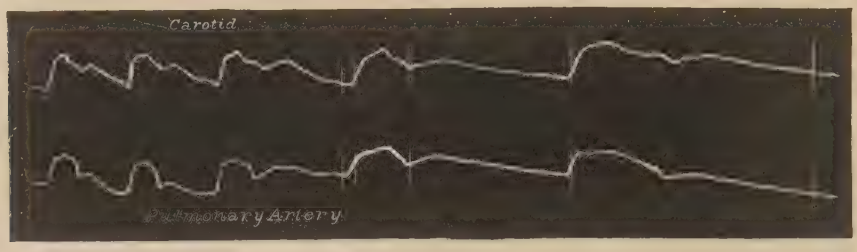


Fig. 295. Simultaneous tracings of the pulses of the carotid and pulmonary arteries. After the third beat the cylinder was rotated rapidly.

exactly over the apex. I shall show later on that this shrinking can be demonstrated in various places, and I have found evidence of it immediately under a diffuse apex beat. The movement producing the apex beat is really a displacement of the heart forward, and while the heart is thus displaced, the ventricles shrink as they empty themselves. The termination of the ventricular outflow is occasioned by the pressure in the aorta becoming higher than that in the ventricle. The semilunar valves close in consequence,

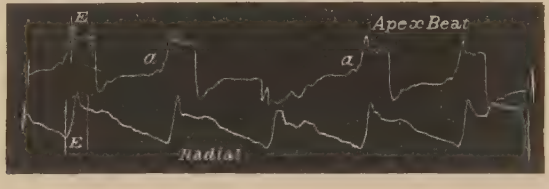


Fig. 296. Simultaneous tracings of the apex beat and of the radial pulse, showing the 'systolic plateau' and the small wave (*a*) due to the auricular systole. The third beat in the apex tracing is obliterated by the movement of inspiration.

and the ventricular muscle then relaxes; the termination of the systolic period is indicated in the cardiogram by a sudden descent.

(*c*) *The period of relaxation of the ventricular muscle* (space *F*, Fig. 293). —With the relaxation of the ventricular muscle the apex retreats from the chest-wall, as is indicated by the slanting downstroke in the tracing, or where the tracing is already falling during the ventricular outflow by a more rapid descent (Figs. 293 and 294). During this period the ventricular pressure rapidly falls until the stage of complete relaxation, when the pressure inside the ventricles becomes lower than that inside the auricles. When this

occurs, the auriculo-ventricular valves open. The apex then has reached its greatest distance from the chest-wall, and in the tracing the lowest point is reached.

The time of the opening of the auriculo-ventricular valves is usually a very definite landmark in apex and jugular tracings, and, in consequence, is a useful standard for measuring the sequence of events in tracings of irregular heart action. It is recognized as the lowest point reached in tracings of the apex of the left ventricle, and it is just before the fall of the wave *v*, in tracings of the jugular pulse. Its time corresponds nearly with the bottom of the aortic notch in tracings of the radial pulse. It is represented by the perpendicular line 6 in many of the tracings given later.

(*d*) *The period of filling of the ventricles* (space *G*, Fig. 293).—Upon the opening of the auriculo-ventricular valves, the blood flows from the auricles into the ventricles; and as the ventricles distend the heart pushes against

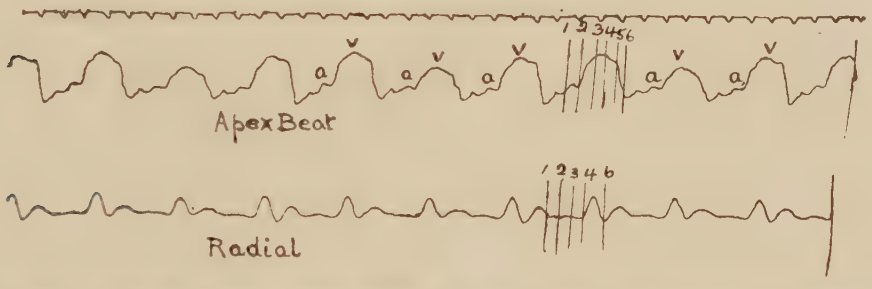


FIG. 297. The apex tracing shows a slight wave (*a*) due to the contraction of the auricles distending the ventricles and beginning one-tenth of a second before the ventricular systole. For explanation of the numbered perpendicular lines see Fig. 320.

the intercostal space, and slightly raises the lever. This period is marked in the tracing by a gradual ascent. Frequently, however, the heart fails to affect the tissues in the interspace during this period, so that no indication of the filling of the ventricles is obtained; in such a tracing as Fig. 292, for example, and in many others given in the text, the whole of this period is a blank so far as information regarding events in the cardiac cycle are concerned.

The auricular wave.—In some tracings from the apex there is occasionally found an abrupt, though slight, rise immediately preceding the beginning of the ventricular systole (*a*, Figs. 296 and 297). This is due to a sudden increase in the contents of the ventricle, caused by the contraction of the auricle, and may be termed the auricular wave.

The auricular wave is not always perceptible in apex tracings, but when present it often gives valuable information. Normally, it precedes the beginning of the wave due to the ventricular systole by about one-tenth of a second (space between 1 and 2, Fig. 297). Sometimes this interval is increased; and then it may indicate a delay in the passage of the stimulus from

auricle to ventricle. In cases of heart-block, it may be recognized during the ventricular pauses. Its absence may be of no significance, but it is to be noted that it is never seen in cases with auricular fibrillation, even when immediately before the starting of this abnormal rhythm it had been a conspicuous feature.

Retraction of yielding structures in the neighbourhood of the heart during ventricular systole.—When the ventricles expel their contents they must of necessity shrink. This shrinkage occurs abruptly, and with considerable force. The yielding tissues in the neighbourhood of the heart are dragged upon, and evidence of this dragging can be obtained from a variety of sources. John Hunter originated the idea that the systole of the ventricles would have a tendency to produce a vacuum, and thus expedite the flow of the venous blood into the chest. Evidence of this 'cardiac aspiration' affecting the lungs has been obtained by a number of observers. The tracings of Mosso and Delépine of the movements of the column of air in the respiratory passages, due to the cardiac aspiration, correspond exactly

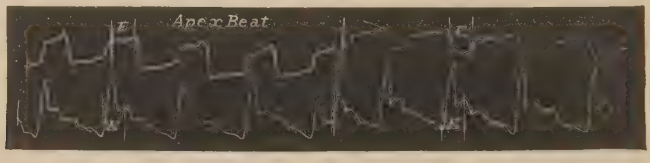


Fig. 298. Simultaneous tracings of the heart movements (upper tracing) and of the radial pulse. The first part of the upper tracing was taken from the apex beat in the fourth interspace immediately outside the nipple, while the latter part was taken in the same interspace near the left border of the sternum. In the first part the cardiogram shows a 'systolic plateau' during the ventricular outflow (*E*); in the other part the cardiogram is inverted, *i.e.* there is a depression during this period (*E*).

with those obtained from the præcordium (Fig. 298), from under the liver (Figs. 299 and 300), and from the epigastrium (Figs. 301 and 302).

In Fig. 298 this drawing-in of tissues in the intercostal spaces over the heart is demonstrated. It was obtained from a boy aged 14. The apex beat was well marked in the fourth interspace outside the nipple. At the same time that the apex was thrust outwards, the skin and subcutaneous tissues over the same interspace inside the nipple were drawn in. In Fig. 298 the tracings of the apex beat were taken simultaneously with the radial pulse for four beats. The clockwork was then stopped, and the receiver, which had been applied over the apex, was placed over the præcordium inside the nipple, and the 'inverted cardiogram' of the last portion was obtained. The space *E* represents the duration of the outflow from the ventricle; and this period, which in the apex tracing shows a flattened elevation, shows a great depression in that obtained from the front of the heart. The ascending limb of the apex tracing corresponds to the period during which the ventricle is contracting (space *D*, Fig. 293). This period in the inverted cardiogram is represented by a slight rise, due to the shock

of the contracting ventricle. No blood as yet has escaped from the ventricle. As soon as the semilunar valves open, the blood rushes out of the ventricle, the ventricles diminish in size, and the yielding tissues of the interspace sink in and cause the great fall, as represented in the inverted cardiogram (space *E*, in the latter half of Fig. 298).

Liver movements due to cardiac aspiration.—Not only can this aspiration be demonstrated as affecting the pliable tissues immediately in contact with the heart, but in suitable cases it can be shown to produce a distinct excursion of the liver. All writers referring to this movement of the liver speak of it as a downward thrust during the ventricular systole. Careful tracings demonstrate that this movement is quite of the opposite nature—

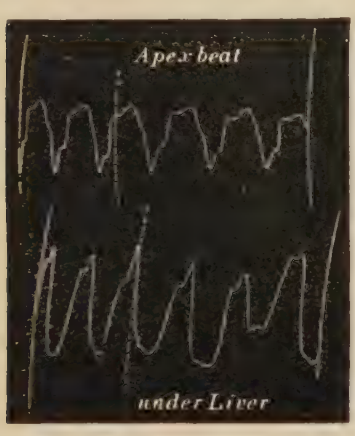


Fig. 299. Simultaneous tracings of the apex beat and of the movement of the liver. When the ventricle empties, the liver is drawn up, and this causes the fall in the tracing.

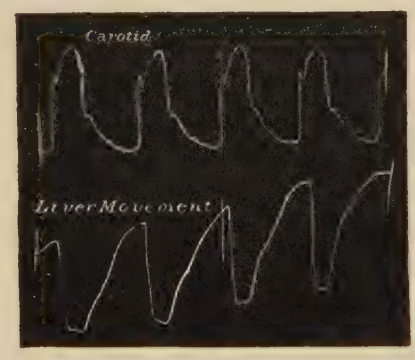


Fig. 300. Simultaneous tracings of the carotid pulse and liver movement. With the appearance of the carotid pulse there is a sudden fall of the lower tracing, due to the liver being drawn upwards by the emptying ventricles.

it is a drawing-up of the liver during the ventricular systole. In Fig. 299 the apex beat is taken at the same time as the movement of the liver.

The receiver, taking the liver movement, being applied to the under surface of the organ, a retraction of the liver upwards corresponds with a fall in the tracing, and vice versa. It will be seen that the movement of the liver upwards takes place during the ventricular systole, while the downward movement is due to the diastolic filling of the ventricle. In Fig. 300 the movement of the liver is recorded at the same time as the carotid pulse. It is seen that as soon as the carotid pulse appears, the liver is drawn up, and remains there until the end of the ventricular systole, after which the liver gradually falls down. I do not mean that the excursion of the liver is one of considerable extent, but the movement is so great as to be obvious to the palpating hand. It is distinct from a pulsation of the liver, which is a periodic swelling of the liver, while this is a displacement of the liver *en masse*.

Epigastric pulsation.—The causes which may produce a pulsation in the epigastrium are : (*a*) a dilated right heart ; (*b*) a hypertrophied left ventricle ; (*c*) the abdominal aorta ; and (*d*) an aneurysm of the abdominal aorta.

In the later stages of typhoid fever and other exhausting diseases, epigastric pulsation is an ominous sign of cardiac enfeeblement. The movement consists of an alternate swelling and retraction of the epigastrium. It is invariably

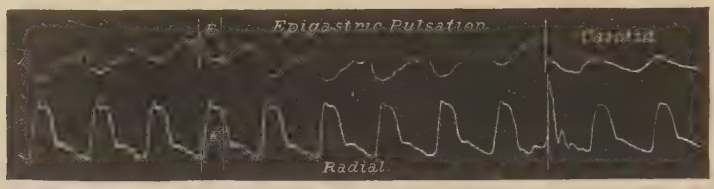


Fig. 301. Simultaneous tracing of the epigastric pulse, due to a dilated right heart, and of the radial pulse. The epigastric pulse shows a retraction during the ventricular systole (*E*), and a protrusion during the filling of the ventricle.

assumed that this swelling or pulsation is due to the right ventricular systole, and that it is of the same nature as the outward protrusion constituting the apex beat. If this form of epigastric pulsation is carefully timed with the carotid pulse, it will be found that the epigastric pulse, protrusion, or swelling precedes the carotid pulse, and that the retraction of the epigastrium corresponds in time to the carotid pulsation. The apex beat is rarely available in these cases, on account of the right heart pushing the left ventricle backwards. In the tracings of the epigastric pulse (Fig. 301) the radial pulse is taken as the standard of time. The time occupied by the pulse travelling from the

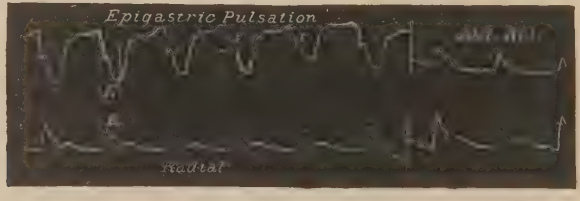


Fig. 302. Shows the same features as Fig. 301, with the exception of the small wave (*c*) occasioned by the shock communicated to the epigastrium by the abdominal aortic pulse. A few beats of the abdominal aorta are also given.

heart to the wrist being allowed for, it will be found that the great fall in the epigastric pulse corresponds exactly with the ventricular systole (*E*).

The patient from whom this tracing was taken was dying from pernicious anæmia. At the post-mortem examination, a needle pushed through the epigastrium, at the place where the tracing was obtained, was found to have penetrated the right ventricle.

In Fig. 302 a similar tracing is given, except that there is a slight interruption at *c*, on the line of descent. This will be found to correspond exactly

to the time of the abdominal aorta, taken from the middle of the abdomen, a few beats of which are also given. This small wave (*c*) is due to the impulse imparted to the tissues by the pulse of the underlying aorta. Epigastric pulsation, due to hypertrophied left ventricle, has the same character as an apex beat (Fig. 303). Epigastric pulsation, due to the abdominal aorta, presents quite a different character from that due to a dilated right ventricle, as shown in Fig. 304, where the pulse corresponds in character and time

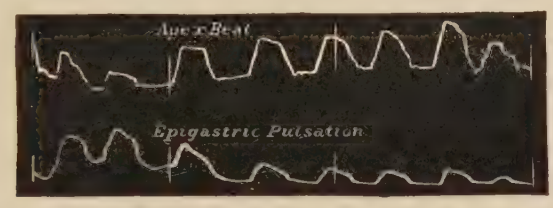


FIG. 303. Simultaneous tracings of the apex beat and of the epigastric pulsation, due to a hypertrophied left ventricle.

with the radial pulse. An epigastric pulsation, due to an aneurysm of the abdominal aorta, would assume the time and character of the abdominal aortic pulse (Fig. 304).

The apex beat due to the right ventricle.—Accepting the usual clinical definition of the apex beat 'being the lowest and outermost part of the heart's impulse', a totally different form of beat is found when the right ventricle causes this movement. In certain cases of dilatation of the right heart, nearly the whole anterior aspect of the heart is composed of the right auricle and ventricle, the left ventricle forming but a mere strip of the border (see Figs. 247 and 248). This portion of the left ventricle is situated so far

FIG. 304. Simultaneous tracings of the epigastric pulse, due to the abdominal aorta, and of the radial pulse.

back that it is covered by the lungs, and does not reach the chest-wall. Hence it is that 'the lowest and outermost part of the heart' in contact with the chest-wall is the right ventricle. The character of the apex beat now corresponds exactly with that of the liver movements, of the epigastric pulse, due to enlarged right heart, and of the inverted cardiogram in Fig. 305. In place of the outward thrust during the systole, as in the apex beat due to the left ventricle, there is an indrawing of the tissues.

Fig. 305 was taken from a youth, aged 18, with simple dilatation of the

heart, and free from valvular disease. There was marked pulsation of the jugular veins, a few beats of which are given. The apex tracing shows a great depression during the period of ventricular outflow (*E*). This period is immediately preceded by an abrupt rise, due to the shock communicated to the chest by the sudden hardening of the ventricular wall. Although corresponding with the period *D* (in Fig. 293) in the left ventricular apex tracings, I am inclined to think that the rise here is an instrumental fault, due to the violent shock communicated by the sudden and forcible ventricular contraction. This period is preceded by a small wave (*a*) in the tracing, identical with the similar rise, in Figs. 296 and 297, of the left ventricular apex beat. It is due in this case, as in those, to the distension of the ventricle by the auricular systole. It occupies exactly the same period in the cardiac revolution as the wave *a* in the venous pulse, which is produced by the systole of the right auricle. The space *E*, in all the tracings, represents the period of ventricular outflow as it affects the different pulses. One can, therefore,

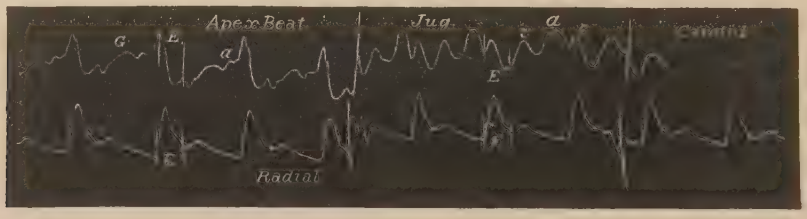


Fig. 305. Tracings of the apex beat, jugular pulse, and carotid pulse (upper tracing), taken at the same time as the radial. The apex tracing is due to the right ventricle, and shows a depression (*E*) during the ventricular outflow. The sharp elevation preceding *E* is caused by the shock of the contracting ventricles. This is preceded by a small wave (*a*), which is due to the contracting auricle distending the ventricle, and corresponds in time exactly with the wave *a* in the jugular pulse, which is due to the contracting auricle sending a wave of blood back into the veins.

readily and with certainty refer the different events to their causes. Thus we know that the wave of contraction arising in the auricle passes on to the ventricle, that between the auricular outflow and the ventricular outflow a period, the presphygmic interval (*D*, Fig. 293), exists during which the ventricle is contracting and raising its pressure until it opens the semilunar valves. Thus the presphygmic period in the apex tracing exactly corresponds to the period between the summit of the wave *a* in the venous pulse, due to the auricular systole, and that of the wave *c*, due to the carotid pulse. A few beats of the carotid are given, which can be taken as a standard of time to verify all these points. The period *G* is due to the filling of the ventricle.

Significance of the inverted cardiogram.—It is asserted in text-books that an indrawing of the apex, during systole, of the ventricles is a diagnostic sign of adherent pericardium. I have had several cases where I have got at one time tracings of the apex beat due to the left ventricle, and at other times tracings due to the right ventricle, with indrawing during systole ; and

at the post-mortem examination there has never been found any signs of adherent pericardium.

The fact that ' the lowest and outermost point of the heart which strikes against the chest-wall ' may be due to the right ventricle should be borne in mind. Whenever that occurs, the cardiogram is an inverted one—that is to say, there is a shrinking of the heart from the chest-wall during the systole, and a protrusion during the diastole of the ventricles. This is not always recognizable at first sight. Being somewhat familiar with the form of various apex-beat curves, I generally have no difficulty in recognizing cardiograms due to the left ventricle. But when from a patient I took Fig. 306 I certainly was misled in the first instance. The abrupt rise and fall bear a close resemblance to an apex-beat curve, due to the left ventricle. Careful measurements of the radial and apex tracings show that the elevation in Fig. 306 was not at the period of ventricular systole, but occurred during ventricular diastole, while the systolic period (*E*) corresponded with the

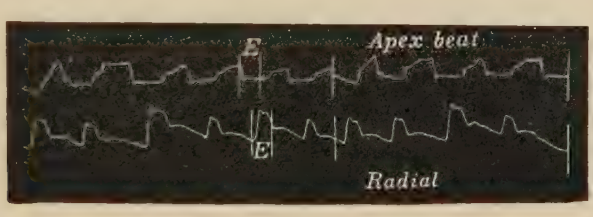


Fig. 306. Simultaneous tracings of the apex beat, and of the radial pulse. The rise in the apex tracing resembles the usual characters present in a tracing of the apex beat due to the left ventricle. On analysis, it is found that the elevation is during the diastole, and the fall (*E*) during the systole, of the ventricle, and therefore the apex beat is due to the right ventricle.

fall in the tracing. It is necessary to insist upon this view, because inferences drawn from the apex beat alone are liable to lead one into error. Even so careful an observer as Keyt has mistaken the nature of such an apex-beat tracing, and imagined in consequence that he detected an extreme delay in the appearance of the arterial pulse. I have observed similar errors of interpretation in the tracings of other writers. It follows, then, that for a guide to any event occurring during a cardiac revolution, the arterial pulse is the only safe and reliable one. When the apex beat is taken as a standard, careful inquiries should be directed to ascertain its true nature. While it is true in the majority of instances that the cardiogram from the right ventricle is ' inverted ', I have taken tracings with a systolic plateau from the third and fourth interspaces near the sternum ; but, as I have had no post-mortem examination in these cases, I am not sure of the part of the heart producing these curves. The whole subject of cardiography is in great need of thorough and painstaking investigation.

Alteration of the apex beat due to retraction of the lung.—If one watches the progress of a case of advancing heart failure over a period of years, marked changes will sometimes be detected, not only in the character,

but in the position of the apex beat. In the earlier stages of heart failure, due to mitral disease, for instance, the left ventricle may be pushed back by the distended right ventricle, so that it is entirely covered by the lung, and the apex beat may then be due to the right ventricle. In course of time, from pressure of the enlarged heart, the lung is compressed and recedes, leaving a large surface of the heart bare to the chest-wall. In such cases the apex beat may be found in the posterior axillary line, and in the eighth interspace. The tracing obtained then is one due to the left ventricle.

The shock due to the ventricular systole.—I am of opinion that a good deal of confusion, in regard to the correct interpretation of the heart movements, has arisen from associating the shock conveyed to the chest-wall when the ventricles pass into systole, with the apex beat. The apex beat and this impulse have become so connected that it is assumed that they are one and the same thing. The apex beat due to the left ventricle is a movement which lasts during the whole of the ventricular systole; the shock caused by the ventricular contraction endures but a short space of time, and occurs while the ventricular muscle suddenly hardens, and corresponds with the upstroke only of the apex beat (*D*, Figs. 293 and 294). It is this shock which sends the lever so high in Figs. 296 and 305, at the beginning of the ventricular contraction. In the tracings of the epigastric pulse (Figs. 301 and 302), and of the movement from the front of the heart (Fig. 298), this shock causes the sharp elevation just before the fall (*E*) due to the emptying of the ventricle. Thus, in noting the time of the shock and watching the epigastric pulse, for instance, as in Figs. 301 and 302, one could see that the retraction of the epigastrium followed it. If one associated the shock with the apex beat, it would therefore be assumed that the protrusion corresponded with the systole, and the retraction with the diastole. It frequently happens that this shock is the only movement of the heart discernible on examining the chest. It is often markedly present in dilatation of the heart, when the heart's surface in contact with the chest-wall is entirely made up of the right ventricle and auricle. In such cases it must not be assumed that the shock is the evidence of the contraction of the right ventricle only. It is impossible to distinguish the shock, due to the right ventricle, from the shock due to the left. The reason I insist upon this is because the perception of this shock has been assumed to be an evidence of the right heart contracting, when the absence of a beat in the radial pulse was supposed to indicate the absence of a contraction in the left ventricle. As will be shown later, this sort of evidence is not only unreliable but actually misleading.

CHAPTER LXVI

Examination of the Arterial Pulse

Superiority of the digital examination.
What is the pulse ?
Inspection of the arteries.
Digital examination of the arteries.
The condition of the walls.
The size of the artery.
Character of the pulse.
The pulse-rate.
The size of the pulse-wave.
The impact of the pulse-wave on the finger.
The rhythm of the pulse.
The two radial pulses compared.
The value of a sphygmogram.
Definition of a sphygmogram.
Events occurring during a cardiac revolution revealed by the sphygmogram.
Features of the sphygmogram due to instrumental defect.

Superiority of the digital examination.—In the examination of the arterial pulse several methods may be employed, as exploration by the finger, by graphic records, and by instrumental measurement of the arterial pressure. By far the most important of these methods is the first. There is a tendency to exalt the others at the expense of the digital, but no apparatus can ever replace the trained finger. No doubt the other methods can give very definite information of a limited kind, but in diagnosing the patient's condition they should only supplement the digital examination.

The mechanical methods can be of use, however, in enabling us to appreciate the meaning of the sensations felt by the finger ; and the attempt should always be made to correlate these sensations with the results obtained by the more elaborate means.

Warning must be given against estimating the patient's condition by the study of the pulse alone ; any definite result obtained must only be employed as one of a group of symptoms on which the ultimate opinion is based.

What is the pulse ?— In order fully to appreciate the study of the arterial pulse, it is essential to have a proper conception of the true nature of what it is we perceive when we examine the pulse with the finger. Broadbent very properly calls attention to a universal misconception of what the pulse is, and points out that it is not an expansion of the artery

due to the blood discharged into the aorta. Marey says that the expansion is so slight that many physiologists have denied its existence, and he states that Poiseuille has demonstrated that in the larger arteries a slight expansion with each systole does take place. No doubt the aorta and its primary branches are somewhat dilated by the injected blood; but whatever the expansion may be in them, in the carotid and radial it must be very minute. To feel the pulse or to take a tracing, it is necessary that the artery should be flattened against the bone. Lister states that it is for this reason that surgeons, operating in close proximity to a large artery, may be utterly unconscious of its neighbourhood, unless they inadvertently wound it, or recognize its pulsation by having compressed it against some resistant structure. The visible movements of the artery are extremely deceptive. They often give the appearance of contracting and expanding; but if the movement be critically examined, it will be found to be, in reality, a displacement of the artery. A straight artery, like the carotid, resembles somewhat a cord that is periodically tightened and slightly relaxed. During the systole of

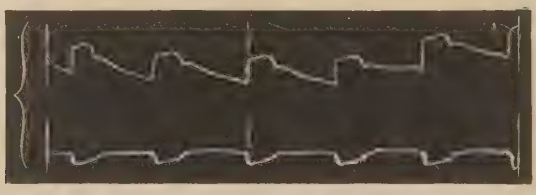


Fig. 307. The upper tracing was taken with the receiver over the carotid artery, at the same time as the lower one was taken with the receiver placed by the side of the carotid artery. The lower tracing is the inverse of the upper.

the ventricle, the carotid is straightened and tightened, and it becomes slightly relaxed during the ventricular diastole. In persons with thin necks, this movement can be studied. If we place one receiver over the carotid and one alongside it, and have the movements properly registered, the one tracing will be found to be the exact reverse of the other (Fig. 307). If the artery expanded during the ventricular systole, it would naturally thrust out all the tissues surrounding it, and the tracing from the side would then be an exact duplicate of the one taken from the front of the artery. The movement, then, of the beating carotid is one of displacement of the whole vessel, not a dilatation and contraction of the vessel.

A similar confusion arises in studying an artery when it is tortuous. In looking at the radial when it is tortuous, one can readily imagine that the rising and falling of the artery is really a distension and contraction of the artery. But if a suitable case be taken, where, in the course of the tortuous artery, there is a short lateral bend, the movement can be demonstrated to be due to the displacement of the artery, and not due to expansion and contraction of the artery. If the pad of the sphygmograph spring be placed close to the artery on the concave side of the bend, and a tracing taken,

it will be found that during the ventricular systole the bend is exaggerated, the artery being pushed farther away from the straight course, and during ventricular diastole the bend diminishes. If tracings be taken of the pulse in such a radial artery at the same time as the carotid pulse (Fig. 308), it will be found that the radial gives an inverted tracing, comparable to that in the preceding figure. If the visible movement were due to the expansion and contraction of the artery, the lever would, on the contrary, rise during systole and fall during diastole, as in an ordinary sphygmogram.

What we recognize, then, as the pulse is the sudden increase of pressure within the artery pressing against our finger, when we compress the artery. With the cessation of the ventricular systole the resistance to our finger steadily diminishes, until the next ventricular systole suddenly rises. Broadbent uses the following apposite illustration: 'Such a pulsation can be felt on a large scale by placing the foot on the inelastic leather hose of a fire-engine in action, in which there can be no expansion'.

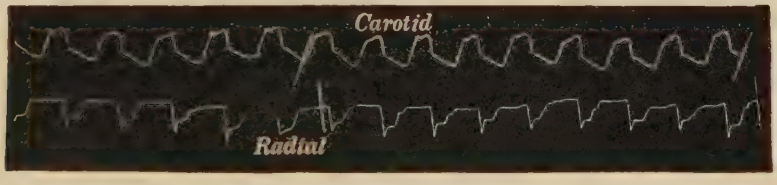


FIG. 308. Simultaneous tracings of the carotid and radial pulses. The radial tracing was taken by placing the pad of the sphygmograph by the concave side of a bend of the tortuous radial artery. During systole, the artery receded from the sphygmograph, and returned during diastole, and hence the tracing obtained is an 'inverted sphygmogram'.

To speak of the pulse as being the expansion and contraction of the arterial walls, or 'the swinging backwards and forwards of the arterial wall', is not only to use language of exaggeration, but to convey a totally erroneous conception of what the pulse really is.

Inspection of the arteries. — Inspection of the arteries reveals in health but little movement. Conditions giving rise to forcible action of the left ventricle may render the pulse visible in some of the superficial arteries. Exertion, excitement, or the febrile state may induce visible beating of the carotids, while this is a marked feature when the arteries are tortuous and atheromatous, and in such a disease as exophthalmic goitre. In free aortic regurgitation, not only is there marked pulsation of the carotids, but pulsation is visible in numerous superficial arteries in various situations. The tortuous character of superficial arteries is visible in arterial degeneration.

Digital examination of the arteries. — It is usual, in the routine examination of the pulse, to place two or three finger-tips on the radial artery near the wrist. The fingers are laid on the artery, and moved upwards

and downwards and across the artery, at first gently, and then with more pressure. By this procedure a knowledge of the size of the artery and the condition of its walls is acquired. Steady pressure being applied in order to obliterate the pulse, the force required to attain this gives an idea of the arterial pressure, and of the character of each individual pulse-wave. It is a good thing to practise the digital examination of the pulse and to compare it with sphygmographic tracings, taken at the same time. By this means the character of the pulse will be better appreciated by the finger.

The condition of the walls.—We recognize the yielding nature of the arterial coats in healthy arteries. In degeneration of the coats the arterial walls may be universally thickened, or contain bead-like patches of induration, as in atheroma, or the artery may have become a rigid tube, as in calcareous degeneration.

The size of the artery.—The variations in size depend entirely upon the degree of relaxation of the muscular coat of the artery. A large artery is not necessarily significant of a strong pulse, nor a small artery of a weak pulse. An increase in the size of the artery frequently implies diminished opposition to the work of the heart. The size of the artery can sometimes be readily appreciated by lightly rolling it under the fingers. At other times it can only be detected, when the pulse is elicited by firm pressure at the place where we expect to find it. This difficulty may occur where there is a good-sized artery embedded in a fat, well-padded wrist, or where the artery is small and contracted. On the subsidence of a fever, a notable diminution in the size of the artery can often be readily recognized.

Character of the pulse.—The trained finger is as yet the best guide we have in judging the character of the blood-stream within an artery. The knowledge necessary to determine what is normal, and what is abnormal, can only be acquired by the constant study of the pulse. The finger-tips become so educated in course of time that we readily appreciate the sensation conveyed in compressing an artery.

The pulse-rate.—The reckoning of the pulse-rate should be made at a late stage in the examination. When abnormally rapid, it should be again counted when the patient has regained his composure. It is best enumerated in two separate half-minutes, to ascertain if the heart is acting quite steadily. In children, unless asleep, abnormal frequency is often very unreliable as a guide, as the presence of the doctor often keeps up a continued excitation of the heart. While the rate of the pulse normally indicates the number of the contractions of the left ventricle, it sometimes happens that these are so weak that some of the pulse-waves are not perceptible to the fingers. In such cases the pulse is usually slow or irregular in rhythm. To appreciate the significance of the pulse-rate, due regard should be paid to the age and idiosyncrasies of the patient, and to the ailment from which he suffers.

The size of the pulse-wave.—The trained finger can recognize a great variety in the apparent volume of the wave itself. Some waves seem to

roll up under the finger, passing gradually away, while others pass quickly, giving a mere flick to the finger.

The impact of the pulse-wave on the finger.—This may be quick and abrupt, and the pulse-wave quickly disappear (pulsus celer), or the impact may approach the finger gradually and gradually subside (pulsus tardus). Although the pulse-wave occupies such a short space of time, yet the sensitive finger readily recognizes these different features.

The rhythm of the pulse.—The beats usually follow one another at regular intervals, and should be of equal strength. The divergences from the normal rhythm are numerous, and the usual terms employed to distinguish them are, in my opinion, both unsatisfactory and misleading; but this subject has already been fully entered into. In estimating the rhythm of the pulse, one's whole attention should be concentrated upon the observation. If one does not exclude other thoughts from the mind, a variation in the pulse-rate and strength may apparently be felt. This is due to a failure to appreciate the pulse, during a remission of the attention. I have not only been conscious of this myself, but in cases where it was important to note the fact, as in pneumonia, I have found my colleagues describing irregularity as being present, when careful examination revealed a perfectly regular pulse.

The two radial pulses compared.—Finally, the two radial pulses should be compared, and any difference in the character of the beats noted. A difference in the strength of the two pulses may be due either to an abnormal distribution of the arteries on one side or to an interference with the lumen of a vessel on one side. A difference in the character of the pulse usually occurs only in the latter case. The two most frequent conditions altering the character of the pulse on one side are the presence of an aneurysm or an obstruction such as an atheromatous plate, diminishing the lumen of the vessel, on the proximal side of the place where the pulse is examined. When an aneurysm is the cause, the difference is said to be due to a delay in the pulse-wave on one side. There is no delay—the wave is only smaller on the side affected. It is always the left radial that is smaller in aortic aneurysm, for the reason that it only occurs when the aneurysm affects the aorta after the innominate artery is given off, while the aneurysm embraces the region of the subclavian artery.

The value of a sphygmogram.—Although the sphygmogram represents the variations in arterial pressure, and although it can give information in this respect, yet there are so many sources of error that it cannot be trusted implicitly. Its greatest service is in giving an accurate record of the movements of the left ventricle. However eloquent may be the words of a writer, he cannot in a page convey as clear an idea of the rhythm of a heart as a simple pulse-tracing; and if writers had given us more pulse-tracings, their works would have been greatly enhanced in value. It is because it gives us a permanent and accurate record, that a tracing of the arterial pulse is of such great value. When we seek to find the nature of

any movement of the circulation by recording it graphically, the arterial pulse is the best and most useful standard, by which we can find its position in the cardiac cycle, as will be shown later.

Definition of a sphygmogram.—When the spring of a sphygmograph is so accurately adjusted on an artery that it does not obliterate the artery when the arterial pressure is at the lowest, and still slightly compresses the artery when the arterial pressure is at the highest, the spring will oscillate with each variation of pressure within the vessel. This oscillation being communicated to the lever and recorded on the tracing-paper gives us a series of wavy lines, which represent the variations of the pressure within the artery. A sphygmogram may, therefore, be defined as a diagrammatic representation of the variations of pressure within an artery. If we knew exactly the amount of pressure exercised by the spring, we should be able to obtain the value of each movement. But the possibilities of error are so numerous that it is useless to draw conclusions from the amount of pressure supposed to be exercised. From the examination of a tracing we

Fig. 309. Sphygmogram of the radial pulse. The space *E* is the period of ventricular systole when the aortic valves are open; the space *G* the period of ventricular diastole; *s* is the pulse-wave due to the ventricular systole; *n* the aortic notch; *d* the dicrotic wave; and *p* a wave due to instrumental defect.

obtain information on three different points: first, concerning the rate and rhythm of the heart's action; second, concerning the sequence of certain events occurring in a cardiac revolution; third, concerning the character of the blood-pressure within the artery.

Events occurring during a cardiac revolution, revealed by the sphygmogram (see diagrams, Figs. 317 and 320). (*a*) *The systolic period.*—If we take a sphygmogram, we can divide the cardiac cycle into two periods: one (*E*, Fig. 309) during which the aortic valves are open, and the ventricle pours its contents into the aorta; and another (*G*, Fig. 309) during which the aortic valves are closed, and the ventricle is in diastole. For the sake of convenience in describing sphygmograms, these two periods will be referred to as the systolic and diastolic periods, although in the space *G* the presphygmic and postsphygmic periods of the ventricular systole are included (Fig. 317). The character of the systolic portion varies very much in different individuals. These variations depend mainly on the amount of resistance offered by the arteries to the ventricular systole. In such a tracing as Fig. 309, there is first an abrupt rise (*p*), then a fall followed by a continuation of the wave *s* at about the same level. This period is

usually described as being divided into two, the abrupt rise being spoken of as the primary or percussion wave, and the latter portion as the tidal or predicrotic wave (the papillary wave and outflow remainder wave of Roy and Adami). This division has led to the idea that these represent two different events in the pulse itself. As a matter of fact, the abrupt rise *p* above the level of the wave *s* is due to instrumental defect, and the whole period *E* is occupied by the ventricular pressure forcing blood into the arterial system, and corresponds with the period *E* in Fig. 317. In cases where the arterial pressure is low relatively to the strength of the ventricular systole, these two waves are so blended together that so-called percussion and tidal waves can no longer be differentiated (Fig. 310). The whole of this period *E* in the tracing will hereafter be referred to as the systolic period, and the wave *s* as the systolic wave, as it represents the period of ventricular systole, when the ventricle and arterial system are in free communication.

(*b*) *The diastolic period.*—With the closure of the aortic valves, the arterial pressure falls rapidly to the bottom of the aortic notch (*n*, Figs. 309 and 310).

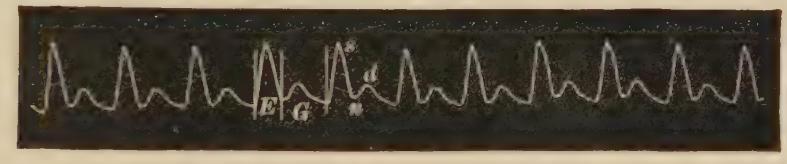


Fig. 310. The letters have the same significance as in Fig. 309.

In the tracings this is seen to be at the beginning of the diastolic period. This fall is interrupted by a distinct rise in the pressure represented by the dicrotic wave *d*. There has been a good deal of discussion concerning the cause of the dicrotic wave. The following explanation seems to me the most probable. The semilunar valves are so delicately constructed that they readily respond when the pressure on one side rises above that on the other. As soon as the aortic pressure rises above the ventricular the valves close. At the moment this happens, the valves are supported by the hard, contracted ventricular walls. The withdrawal of the support, by the sudden relaxation of these walls, will tend to produce a negative pressure wave in the arterial system. But this negative wave is stopped by the sudden stretching of the aortic valves, which, on losing their firm support, have now themselves to bear the resistance of the arterial pressure. This sudden checking of the negative wave starts a second positive wave, which is propagated through the arterial system as the dicrotic wave. After the dicrotic wave, the arterial pressure-curve gradually falls. Occasionally there are slight waves in the fall, but these are of doubtful import.

Features of the sphygmogram due to instrumental defect.—In the study of sphygmographic tracings, one has always to bear in mind that certain

features may be due to the instrument itself. Speaking generally, these instrumental features occur where there are sudden and forcible changes in the arterial pressure. The most frequent of these is the jerking up of the spring by the systolic wave itself. Then the next most frequent is the forma-

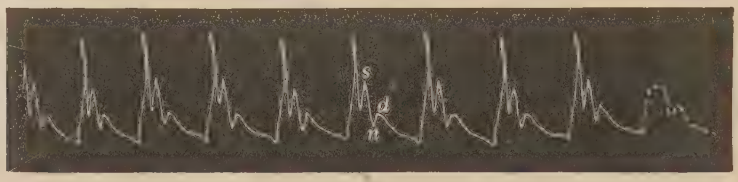


Fig. 311. A strong-beating ventricle has jerked the lever high above the true systolic wave, and the falling lever has made an artificial notch on the systolic wave *s*. The true pulse-curve is probably represented in the dotted tracing.

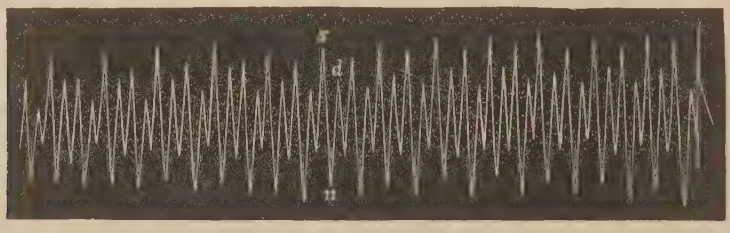


Fig. 312. The forcible changes in pressure have exaggerated the height and depth of all the waves.

tion of a notch on the tracing of the systolic wave, due to the sudden fall of the spring after being jerked high up, as in Fig. 311. Occasionally one finds the aortic notch artificially deepened by the sudden lowering of the pressure, as in Fig. 312.

CHAPTER LXVII

Arterial Pressure

The cause of arterial pressure.
Methods of measuring the blood-pressure.
Increased blood-pressure.
Hyperpiesis.
Effect on the heart of increased peripheral resistance.
Increased arterial pressure and heart failure.
Prognosis of high blood-pressure.
Treatment of high arterial pressure.

The cause of arterial pressure.—When the left ventricle contracts it drives the blood into the arterial system. The escape through the arterioles and capillaries is retarded, so that the blood continues to flow after the ventricle has ceased to contract. As a consequence of this, the arteries are slightly distended during ventricular systole, and their elastic coats compress the column of blood within them after the ventricular systole is over, and thus maintain a degree of arterial pressure during the period in which the ventricle is not acting. The ventricular force is thus stored up by the distension of the elastic coats of the arteries, and liberated during the ventricular diastole.

The chief factors, therefore, concerned in the maintenance of arterial pressure are the ventricular systole, the peripheral resistance, and the elastic recoil of the arteries. Though the factors may be recognized, we are so profoundly ignorant of the part each plays and how they are influenced, that all explanations of the variation of pressure are mere conjectures.

Methods of measuring the blood-pressure.[1]—Of late years many instruments have been devised to measure the arterial pressure. The majority are constructed on the principle of compressing the brachial artery with an air-bag embracing the upper arm. Air is pumped into the bag, and its pressure is measured by a mercury manometer in connexion with it. When the pressure is raised sufficiently to obliterate the radial pulse, we obtain the only really trustworthy standard, and it is this I refer to hereafter as ' arterial or blood-pressure '. In place of using the finger a sphygmogram may be taken, and the

[1] There is no use trying to get an accurate record of the blood-pressure in cases of continuous irregular action of the heart, such as auricular fibrillation. The size of the beats is always varying, and any attempt to gain even an approximate figure to represent the blood-pressure is little more than guesswork, so that observations based on such results are not only unreliable but misleading.

pressure noted when the pulse-beats fail to move the lever (see Figs. 313 and 314).

Another method is to auscultate the artery at the bend of the elbow. When the pressure is raised in the cuff to 50 or 60 mm. Hg. the artery is partly compressed, and a slight sound is heard, which increases in loudness while the pressure is being raised, till it fades away with the complete obliteration of the lumen of the artery. The best way is to raise the pressure rapidly till

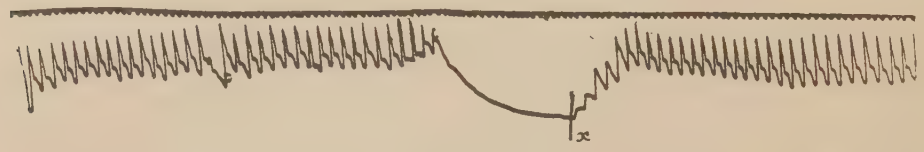


Fig. 313. The clockwork of the sphygmograph was slowed and the blood-pressure taken. The pressure in the arm cuff was raised till the pulse ceased. The air was allowed to leak slowly and the height of the mercury noted at *x* (158 mm. Hg) on the appearance of the first pulse-wave.

no sound is heard, then gradually allow the air to escape from the cuff, until the sound returns. The pressure at the first appearance of the sound is noted as the 'systolic pressure'. The pressure on the cuff is allowed to fall, and after a time the sound is heard to alter its character, becoming much softer. This period is, according to MacWilliam, the 'diastolic pressure'. The sound may persist till its pressure falls still lower, and in some cases of aortic regurgitation the sound may be heard when the cuff is empty.

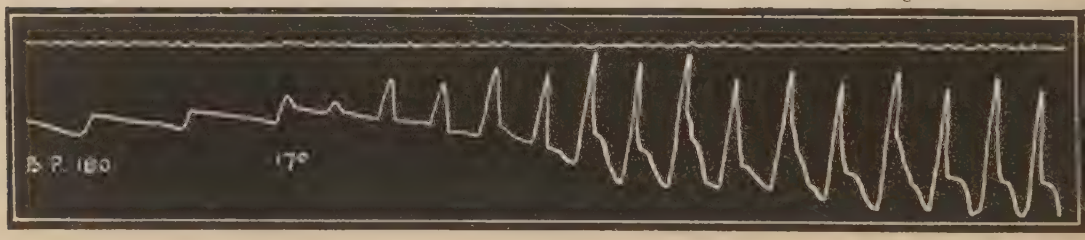


Fig. 314. Tracing taken from the radial artery after the air-bag connected with the manometer had obliterated the pulse at a pressure of 190 mm. Hg. The air was allowed to escape gradually, and when the pressure fell to 180 mm. Hg the sphygmograph received the stronger beats, and the smaller beats came through when the pressure fell to 170 mm. Hg. When the pressure in the air-bag was exhausted the pulse tracing showed rhythmical variation in the size of the waves—that is, the pulsus alternans.

Attempts have been made to estimate the systolic, mean, and diastolic pressures by observing or recording the movements communicated to the column of mercury by the compressed artery. It is found that, during the gradual compression of the artery, oscillations due to the pulse-beat occur in the mercury. These oscillations begin, gradually reach a maximum, and gradually decrease as the pressure is raised or lowered. Far-reaching deductions have been drawn from the changes in these oscillations. As the cause

of these oscillations is not understood, the attempts to estimate the mean or diastolic pressure from them is little better than guesswork, and in practice it will more likely tend to mislead than to give reliable information (see diagram, Fig. 315).

The force required to obliterate the pulsation in the radial artery is fairly easily ascertained, and from it certain limited inferences may be drawn. It is doubtful if it represents the actual arterial pressure within the artery, for an escape can take place imperceptible to the finger, and certain external conditions may affect the pressure. It is usually assumed, for instance, that the arterial wall and its coverings offer such a slight resistance as to be negligible. Russell, on the other hand, asserts that the thickening or contraction of the artery may have a very considerable effect, and that thick, sclerosed, and contracted arteries may offer such resistance that a considerable proportion of the pressure may be spent in overcoming it. Oliver has shown that the pressure obtained by instruments may vary in different

Fig. 315. Diagram to show the nature of the oscillations of the mercurial column in estimating the blood-pressure. *A* and *B* represent the walls of the brachial artery. At 160 the lumen of the artery is obliterated by the pressure of an air-bag embracing the upper arm. As the pressure is gradually lowered in the bag, each beat of the artery produces a movement of the mercury in the manometer, the movement being represented by the elevations in the diagram. With diminution of pressure there is at first a gradual increase in the extent of the movement, followed by a gradual decrease until the pressure in the air-bag ceases to compress the artery. From this it is shown that there is no definite period which can be said to correspond to the systolic, diastolic, or mean blood-pressure.

arteries in the same individual. Hill, Rowlands, and others have shown that in aortic regurgitation the pressure in the femoral artery may be much higher than in the brachial, as much as 100 mm. Hg. By immersing the trunk in hot water, these differences disappear. This remarkable difference is probably due to some modification in the arterial wall, but its nature I do not understand.

There are a great many different instruments devised to take the arterial pressure. Most of them are somewhat cumbrous, and some patients resent the disagreeable sensation produced by compressing the upper arm. Hence these methods are not likely to receive that general application which the need for ascertaining the blood-pressure requires. L. Hill has recently invented an extremely simple and practical method. In place of a cuff surrounding the upper arm, he uses a small bag, which is compressed over

the radial artery, until the pulse disappears below the place of compression. The bag is connected with a very simple manometer, which can be carried in the waistcoat pocket.

A great deal has been written about blood-pressure and its estimation in practical medicine; but it must be confessed that much of it has been of little practical value, and much careful observation, extending over many years, on individual patients, will be necessary before any sure and certain result is obtained. The remarks I make on the subject are based on the examination of numerous patients, in the endeavour to ascertain some sure foundation on which to base the application of the method in the clinical examination of heart affection.

Increased blood-pressure.—In many cases one can corroborate by instrumental observation the knowledge previously acquired by the finger, that the pressure in the arteries increases in certain diseased conditions, as Bright's disease, and with advancing years. As the increased pressure of advancing years is associated with arterial changes, the question of cause and effect is a very difficult one to solve. On the one hand, the changes in the blood-vessels undoubtedly tend to raise the blood-pressure, while it is contended that these are induced by the blood which contains ingredients that provoke a contraction of the arterioles, in consequence of which the muscular coat hypertrophies. A rise in pressure seems to induce atheromatous degeneration, and this in turn causes a rise in pressure. The fact is undoubted, that arterial changes and high blood-pressure are very frequent phenomena in advanced life. The changes are so insidious that they rarely come under consideration until they are well established. One may infer, indeed, that such changes are occurring in middle life, when one notices a tortuous radial artery, but it is rare that the condition gives any cause for anxiety until it is well established.

It is a mistake, and one made not infrequently, to consider the high blood-pressure as if it were a disease. Happily the efforts employed to reduce the blood-pressure are usually of little effect. In order to appreciate the meaning of high blood-pressure, it is well to consider the condition associated with its production, for I think it has a significance beyond that of being a manifestation of disease.

I have already pointed out that the arterial pressure is maintained chiefly by the force of the left ventricle, the peripheral resistance, and the elastic recoil of the arteries. The necessity for the pressure is the regular and equable supply of blood to the organs and tissues. Between the heart and tissues there is an intimate association, whereby the supply to the tissues and organs is moderated by their requirements—the heart beating more forcibly and more rapidly, and the peripheral resistance diminishing, when there is an urgent need, on account of the increased functions of the organs. With advancing age, three great changes occur in the blood-vessels. The elasticity of the arteries diminishes. The result of this is that there is no longer the same equable maintenance of the pressure during diastole. The

arteries approximate the condition of rigid tubes, where the force exerted by the left ventricle is not sufficiently stored up in the elastic coats, to be liberated during diastole. The loss of this assistance necessitates increased force of the ventricular contraction, and, therefore, an increase of the pressure during ventricular systole. In the arterioles there may be an increase of the muscular tissue, and this implies an increase in functional activity with an increase of the peripheral resistance. These are the two factors that are generally assumed to be the cause of increased blood-pressure ; but there is a third which has not received that consideration to which it is entitled, namely, the diminution of the capillary field. This can be recognized in various ways, as, for instance, the thinning and wasting of the skin and subcutaneous tissues, and by the absence of oozing in surgical operations on elderly people. The manner in which it raises the blood-pressure is simply by narrowing the passage of outflow.

Hyperpiesis.—In the routine examination of patients, we meet occasionally with some, usually middle-aged, sometimes young, who show considerable fluctuations in blood-pressure. Periods of high arterial pressure (hyperpiesis of Clifford Allbutt) may be associated with some discomfort, as mental dullness, headache, etc. These periods can be cut short by a smart purge, bodily exercise, etc., or they may disappear from no ascertainable cause. It is possible that such periods of high arterial pressure are due to faulty metabolism ; but they will be found to recur in spite of the greatest care in diet. It is said by some that these periods of high blood-pressure are the cause of arterial degeneration, but, with an imperfect knowledge of all the factors, we are not in a position to decide.

Effect on the heart of increased peripheral resistance.—I have mentioned that the connexion between the heart and the tissues is so intimate that the demand from the tissues is responded to by stronger contraction of the heart. When, therefore, one or all of these causes increasing the peripheral resistance are in action, the heart, in order to supply the tissues with blood, has to exert more force in its contraction. It accommodates itself to its ever-increasing burden by calling upon its reserve, and the only evidence that it has more to do is in the limitation of its field of response. One can almost say, when an individual realizes the fact that a hill is not climbed with the ease and comfort with which it used to be done, that already the heart is meeting an increased peripheral resistance, and there is already a slight diminution of its reserve force. This, as we know, is a very gradual and long-continued process, beginning insidiously in the fourth decade of life, and coinciding with the time at which athletes abandon the exercises that call for long and severe exertion.

As the changes that increase the peripheral resistance tend slowly but surely to advance, the work of the heart becomes ever greater, the field of response becomes more limited, till finally the patient's attention is called by some disagreeable sensation to the fact of the great limitation, and so we get the evidence of heart failure.

But this is not all. The changes that have taken place in the arterial walls in the periphery have, at the same time, been affecting the arteries of the heart, with the result that the muscle-fibres are imperfectly nourished and degenerate. It is in these cases of long-standing high blood-pressure that we find the most striking evidence of degenerative changes in the heart muscle, associated with arterial degeneration, and it is wonderful how long a heart extensively degenerated can maintain a high blood-pressure.

Such cases of degenerated arteries and long-continued high blood-pressure end by a degenerated artery giving way and causing cerebral apoplexy, or by failure of the heart. The latter may come about in various ways: by a sudden change in the inception of the heart's contraction—for it is in these cases we frequently find auricular fibrillation—by gradual exhaustion of the heart's strength, often with angina pectoris, and on rare occasions by rupture of the heart.

Increased arterial pressure and heart failure.—From such considerations it will be realized that we may have heart failure without fall of blood-pressure; and this consideration brings clearly into view the fact that heart failure in these cases is primarily a matter of exhaustion of reserve energy. In cases of valvular disease there may be marked failure of the heart with little or no fall in blood-pressure, and recovery may ensue with little or no rise in blood-pressure.

Prognosis of high blood-pressure.—There has been so much nonsense talked and written about high blood-pressure that I am constrained to draw attention to our extreme ignorance of the cause and consequence of raised blood-pressure. That we are totally ignorant of the cause does not prevent many people adopting heroic measures for reducing it, without even considering the wisdom or necessity for so doing. That it may be a physiological process for the benefit of the organism is seldom considered. It would not be exaggerating to say that the present state of our knowledge of this subject is so wanting in trustworthy data as to warrant neither a sound prognosis nor a rational therapy. In some cases it is associated with manifest disease of the arteries or the kidneys, and here a grave prognosis can safely be ventured; but the high blood-pressure forms but one datum, and the older physicians could have given as sound a prognosis before the invention of the instrument for estimating the blood-pressure.

It must be borne in mind that it is only within recent years that observations approaching accuracy have been in use, and that no one has watched individuals for a long enough time to acquire the knowledge of the variations of pressure which accompany a healthy life. When what has been considered an increase has been detected in an individual, he has been subjected to treatment, and if he has continued living as a normal man, this has been attributed to the measures prescribed for his raised blood-pressure. We constantly see individuals proceeding to some spa for treatment for high blood-pressure, and the spa physician puts an individual through some rigid régime, and, finding the pressure fall, imagines that his treatment has

saved the patient from arterio-sclerosis. I have repeatedly seen such cases, and have taken the blood-pressure before and after their visit to the spa, and have detected no difference, notwithstanding that they have brought back a letter from the spa physician telling of the wonderful way the blood-pressure has been reduced. One constantly meets with individuals who have been under continued treatment for years, because they have had a blood-pressure supposed to be high. The inefficiency of treatment to reduce the pressure has produced in them a state of depression at times approaching panic.

I give here the grounds I have employed for a prognosis in these cases; grounds which are no doubt slight and inefficient, but, so far as I have tested them, of quite a distinct value.

Although I had been studying blood-pressure for years, and used various instruments, it was only in 1905 that I obtained an instrument of some reliability, namely, the Martin modification of the Riva-Rocci. I used only the method by which the disappearance of the radial pulse is detected, when the pressure in the armlet obliterates the pulse—what is called the systolic pressure—though I am not sure that it actually represents the systolic pressure—nevertheless it is a safe standard from which to measure variations. I took records of a good many people whom I could keep under observation. In some I found the pressure vary remarkably. At some examinations it would be 180 mm., a few days later it would be 120. I could find no cause for such variations. In some liable to attacks of angina pectoris the liability was as great when the blood-pressure was low as when it was high. Some with high blood-pressure got heart failure, or apoplexy, and died; others lived and pursued their occupations with no limitation for the years I have been able to watch them. When I came to inquire into the difference between these various types, I found that the former were always associated with evidences of disease of the heart, kidneys, or blood-vessels, of such an extreme degree that a sure prognosis could have been given apart from the blood-pressure. On the other hand, those who pursued a normal life presented no changes in other organs, beyond what one would consider compatible with their time of life. In giving a prognosis, therefore, I am accustomed to review critically the condition of the kidneys, the state of the arteries (inquiring if there has been any hæmorrhage), and the condition of the heart, with particular reference to its size and functional efficiency. It is on the result of such a review, with the knowledge of the blood-pressure, that I base a prognosis: that is to say, if the size of the heart is within normal limits and the heart's power not restricted, then the prognosis is favourable; and the less favourable prognosis is based on the limitation of the heart's power—which is usually associated with an increase in the heart's size.

Treatment of high arterial pressure.—In a long series of observations on the effects of drugs and diet which my colleagues and I have carried on at the London and Mount Vernon Hospitals, we have been struck with

the futility of the methods and drugs usually recommended to lower blood-pressure. Such drugs as the nitrites have only a transient effect, when they do affect the blood-pressure. Many of the observations with the nitrites have been made on the young with healthy arteries, and the conclusions drawn from them have been applied in the treatment of the senile with degenerated arteries. As a matter of fact, we found that in the elderly the blood-pressure was often quite unaffected by fairly large doses of the nitrites.

When there is heart failure aggravated by high pressure, the best line of treatment is to ease the load and give the heart rest, to regain some store of reserve force. There is no use in trying to reduce the blood-pressure by administering drugs of the vaso-dilator class. Luckily, the administration of these drugs is of little effect, and little or no permanent lowering can be obtained by their use. It is manifest that with changed arteries, and a diminished capillary outflow through obliteration of the capillary vessels, a high blood-pressure is necessary to supply the organism with blood. If it were possible to reduce the blood-pressure permanently in a man who for years had a blood-pressure of 180-200 mm., the result would be impaired nutrition of the organism. If the final breakdown of these patients be watched, it will not infrequently be found that the blood-pressure does fall to 150 or 140 mm. Hg, and the result is at once the appearance of the signs of extreme heart failure—dropsy, enlarged liver, œdema of the lungs, etc. So serious is the significance of a fall of blood-pressure in patients with degenerated heart muscle, even with attacks of angina pectoris, that the persistent fall of pressure is an evidence of the final exhaustion of the heart, though the anginal attacks may cease.

The principle I pursue with most success is to place the patient under conditions that give the heart less work to do, carefully avoiding ' cardiac tonics ' and ' vaso-dilators ', restricting the diet, evacuating the bowels, and permitting such exercise as the patient can undertake without distress, according to the lines laid down in the chapter on treatment (Chapter LXII.).

CHAPTER LXVIII

The Venous Pulse

What the venous pulse shows.
Inspection of the jugular pulse.
Methods of recording the jugular pulse.
The recognition of the events in a jugular pulse.
Description of the events in a cardiac cycle.
The causes of the variation of pressure in the auricle and in the jugular vein.
Standards for interpreting a jugular pulse.
The carotid wave.
The notch on the ventricular wave.
The diastolic wave.
Changes due to variation in the rate of the heart.
Method of analysing a tracing.
The ventricular form of the venous pulse.
Anomalous forms of the venous pulse.
Conditions giving rise to a venous pulse.

What the venous pulse shows. — The consideration of the circulation has so far been mainly concerned with the effects of the contraction of the left ventricle. When the apex beat is studied, or the characters of the arterial pulse analysed, our purview is limited almost entirely to the doings of the left ventricle. The arterial pulse, indeed, gives us a direct knowledge of the left ventricle's action during but a portion of the cardiac cycle, namely, during the period when the aortic valves are open. When they are closed we are no longer directly cognizant of what is happening in the left ventricle. We come now to the study of a subject which gives far more information of what is actually going on within the chambers of the heart. In the venous pulse we have often the direct means of observing the effects of the systole and diastole of the right auricle, and of the systole and diastole of the right ventricle. The venous pulse, therefore, presents a greater variety of features, and may manifest variations due to disease which the study of the arterial pulse fails to reveal.

Inspection of the jugular pulse.—In examining a patient for pulsation in the jugular vein, it is generally best that he should lie down, though in some rare cases, where the veins are greatly distended, the pulsation can only be recognized when the patient sits up. The pulsation is most commonly limited to the internal jugular veins, and these veins, lying

alongside the carotid arteries, are never visible, being covered at the root of the neck, not only by the skin and sterno-mastoid muscle, but by a variable quantity of adipose tissue ; one, therefore, recognizes the venous pulse only by the character of the movements communicated to the structures covering the vein. In that form of the venous pulse in which the principal wave is due to the auricular systole, the sudden collapse of the tissues covering the vein is more striking than the protrusion. If one further carefully times this collapse, it will be found to be synchronous with the arterial pulse. The pulse in the internal jugular vein is, in diseased conditions, sometimes mistaken, even by experienced observers, for ' beating of the carotids '. But the carotid pulse is always abrupt and sudden in its protrusion of the covering tissues, and gradual in the shrinking. Furthermore, when one finds a small radial pulse and a large pulsation in the neck, one may safely conclude that the neck pulsation cannot be carotid, unless under very exceptional circumstances (as aneurysm). When the pulsation is in the more superficial veins, as the external jugular, facial, or superficial thoracic veins, the collapse of the vein synchronous with the carotid pulse is usually easy of recognition. In another form of venous pulse, where the pulsation is due to the ventricular systole, the engorgement of the veins is usually so great, the arterial pulse so small, and the cardiac mischief so evident, that the recognition of the venous pulse is comparatively easy.

Methods of recording the jugular pulse. — Usually, the movements of the vein are best recorded with the patient lying down, the shoulders slightly raised, the head comfortably supported by a pillow, and turned slightly to the right in order to relax the right sterno-mastoid muscle. The receiver (*E*, Fig. 286) is placed over the jugular bulb immediately above the inner end of the right clavicle, with just sufficient pressure to shut off the interior of the receiver from the outer air. One may have to shift the receiver about to get the best movement. The relation of the jugular bulb to the surrounding structures is shown in Fig. 316, where the circle above the clavicle indicates the position of the receiver.

Sometimes better tracings are got higher up in the neck, or from the left side. In great engorgement of the veins, it may be possible only to get a tracing when the patient is sitting up. The continued action of the sterno-mastoid in laboured breathing may prevent a tracing of the jugular pulse being obtained.

The recognition of the events in a jugular pulse.—There is still much that is obscure about some of the details of the venous pulse, and several of these are still the subject of controversy. In the following interpretation I deal with the salient points, which have thrown most light upon the obscure features of the heart's action. The movements of the venous pulse are usually more numerous than those of the arterial, and in the tracings a number of waves are present. As each of these indicates a rise of pressure in the veins, the tracing can only be properly interpreted when the force producing each rise of pressure is known ; and for this

purpose the time of appearance of each wave in the cardiac cycle must be established. This is done by taking tracings of the venous pulse, at the same time as some movement whose position in the cardiac cycle is definite, and the arterial pulse, carotid or radial, is the most reliable. The apex beat is often useful and convenient, but care is necessary in the employment of the apex beat, as has already been pointed out.

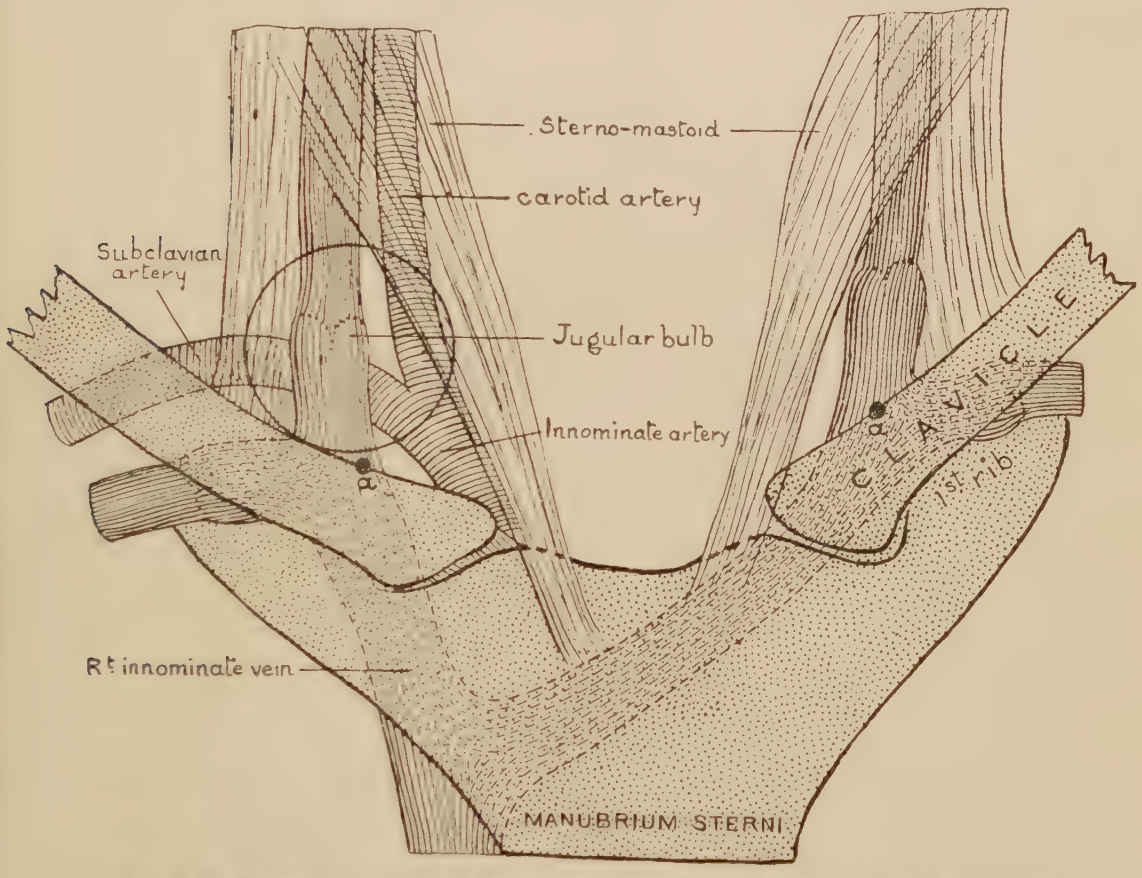


FIG. 316. Shows the relation of the internal jugular vein to the carotid and subclavian arteries, and to the sterno-mastoid muscle. The circle represents the position of the receiver in taking a tracing, and is seen to cover not only the jugular vein but also portions of the carotid and subclavian arteries. The spot at *a* is one inch from the internal end of the clavicle. (Keith.)

Description of the events in a cardiac cycle.—In the diagram (Fig. 317) there is represented a series of movements, due to various forces that occur during one cardiac cycle. If a wave be found in the vein, and if its time of occurrence be ascertained by referring to the place it would occupy in this diagram, we can usually find its cause by noting what force is operative at that period. It must be added that while this diagram represents with

fair accuracy the chief events in a cardiac revolution, it is not asserted that it is correct in every detail. Authorities are not quite agreed on several small points, but it is sufficient for the purpose I have in view.

What we have here presented are, the curves representing the variations of (1) the pressure within the auricle ; (2) the pressure within the ventricle ; (3) the pressure within the aorta. The spaces embraced by the perpendicular lines represent, respectively, the time during which the semilunar valves are open (*E*) and the auriculo-ventricular valves are shut (*D*, *E*, *F*). I would

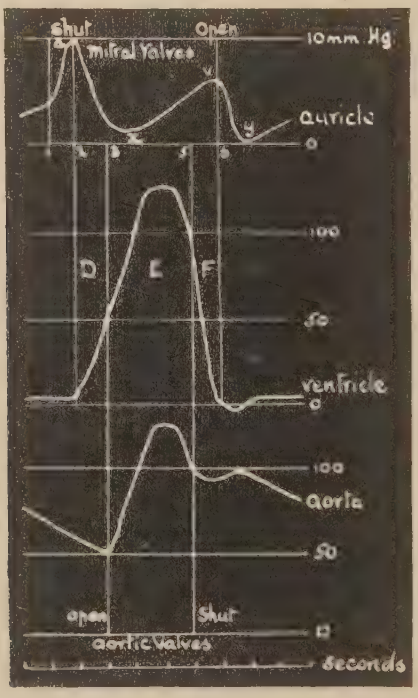


Fig. 317. Semi-diagrammatic representation of the auricular, ventricular, and aortic pressures during one cardiac revolution. *D*, the presphygmic period of the ventricular systole ; *E*, the sphygmic or pulse period ; *F*, the postsphygmic period. The figures 1, 2, 3, 5, and 6 have the same significance as those in Fig. 320. The divisions on the bottom line represent tenths of a second.

direct attention to the presphygmic period *D*, when the ventricular pressure is rising, but has not yet opened the aortic valves ; and to the postsphygmic period *F*, where the ventricular pressure is falling after the closure of the aortic valves. The curves indicating the pressures are approximately correct, but are utilized here to show the periods when variations take place in the pressure. Though the events in the diagram represent what happens in the left side of the heart, there can be no doubt that the changes on the right side are of the same character.

The causes of the variation of pressure in the auricle and in the jugular vein.—The auricular pressure in Fig. 317 shows a series of rises and falls, and these correspond to those in a venous pulse (Fig. 318). The forces operative in producing the variations in the auricular pressure are also acting in producing the jugular pulse. (The curves of auricular pressure given by different physiologists are very perplexing, some getting a rise of varying duration during ventricular systole. I select Frey's as the simplest and probably the truest.)

The auricular wave (*a*) *and fall* (*x*).—The rise *a* in the jugular pulse, Fig. 318, corresponds with the first abrupt rise *a* in the auricular pressure-curve, Fig. 317, and both are due to the systole of the auricle. Neglecting the wave *c* for discussion later, the fall *x* in the jugular pulse corresponds to the fall *x* in the auricular pressure-curve, and occurs at the time the ventricle is in systole. The fall is due to three factors: (1) the relaxation of the auricle

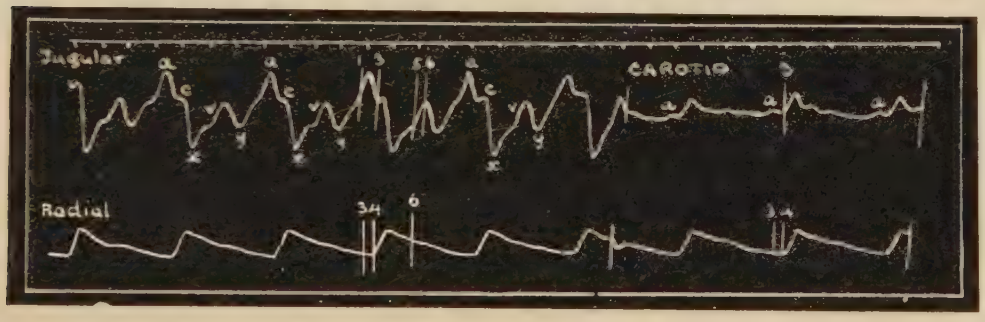


FIG. 318. Simultaneous tracings of the jugular and radial pulses and of the carotid and radial. The elevations *a* and *v*, and falls *x* and *y*, in the jugular tracing correspond to those in the auricular pressure-curve in Fig. 317.

after its systole; (2) the dragging down of the auriculo-ventricular septum by the ventricular muscle, enlarging the auricular cavity; (3) the diminished intrathoracic pressure in consequence of the expulsion from the chest of the contents of the left ventricle. When there is a delay in the ventricular contraction, the factors may be separated as in Fig. 99, p. 171, where *x* is due to the first factor, and *x'* to the second and third factors.

The ventricular wave (*v*).—The rise *v*, after the fall *x* (Fig. 318), is due to the storing of blood in the auricle during the time of the ventricular systole, and corresponds with the second rise (*v*) in the auricular pressure-curve in Fig. 317. The termination of this rise in both figures is sudden, and due to the opening of the auriculo-ventricular valves. *While the beginning of this rise is very variable, its termination is one of the most certain periods in the cardiac cycle, indicating as it does the time of the opening of the tricuspid valves.* The variableness in its beginning is due to the fact that it owes its origin to the quantity of blood stored in the auricle during ventricular systole; and this varies in individual cases, and also in the same individual, with exertion

and respiration. The blood comes mainly from the periphery, pouring into the auricles through the veins. When the auricle becomes filled, the surplus distends the superior vena cava and jugular veins, and hence appears in the tracing as a wave. Another source is sometimes regurgitation through the tricuspid orifice. It is necessary to bear this in mind, as the failure to recognize how tricuspid regurgitation would be manifested in the venous tracing has led to a total misconception of the meaning and nature of the ventricular form of the venous pulse. It has been assumed that, in tricuspid regurgitation, the blood sent back into the veins would appear in the jugular at the same time as in the carotid. In this assumption, the effects of a dilating auricle between the ventricle and veins has been overlooked; as a matter of fact, what happens is merely an increase in the amount of blood accumulating in the auricle during the ventricular systole, and this causes the appearance of the wave *v* to be somewhat premature. Thus, for

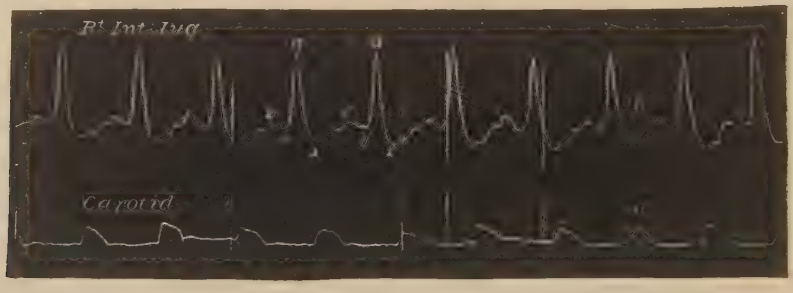


Fig. 319. Simultaneous tracings of jugular and carotid pulses showing that the waves in the jugular correspond in time with the waves of increased auricular pressure in Fig. 317.

instance, Fig. 319 is from a case where there was a damaged tricuspid valve so that regurgitation took place, and the wave *v* is seen to be of small size, beginning early in the time of the ventricular systole (period *E*).

I call this wave *v* the ventricular wave, because of its association with the systole of the right ventricle. Thus the termination of the wave is due to the relaxation of the right ventricle and opening of the tricuspid valves; it is often made up of blood sent back through the incompetent tricuspid orifice by the systole of the right ventricle; though this wave may be small and of brief duration in the auricular form of the venous pulse, it becomes increased in size, and the main or only wave in the ventricular form of the venous pulse.

The fall (*y*), in Figs. 317 and 318, is due to the blood that has been stored in the auricle during ventricular systole, flowing into the ventricle after the opening of the tricuspid valves. Then, as the ventricle becomes filled, stasis in the auricle and veins takes place, causing the rise between *y* and *a* (Figs. 317 and 318), till the auricle again contracts.

Standards for interpreting a jugular pulse.—Such are briefly the main factors concerned in the production of the auricular form of the venous pulse.

It is at times difficult to interpret the tracings, so that it is necessary to have definite standards to help in deciphering certain obscure features.

In Fig. 320 are tracings representing the jugular pulse, the apex beat, and the carotid and radial pulses, to show the relation in time of certain events in these various movements. The numbered perpendicular lines indicate

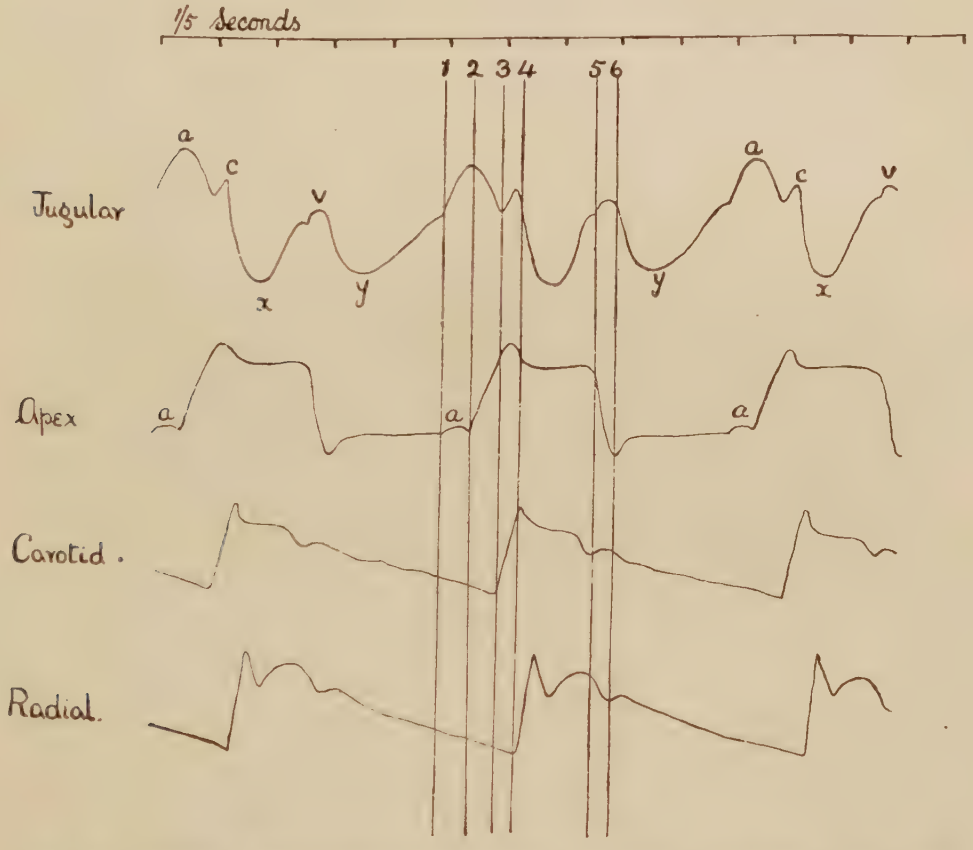


FIG. 320. Tracings of the jugular pulse, apex beat, carotid and radial pulses. The perpendicular lines represent the time of the following events: 1, the beginning of the auricular systole; 2, the beginning of the ventricular systole; 3, the appearance of the pulse in the carotid; 4, the appearance of the pulse in the radial; 5, the closing of the semilunar valves; 6, the opening of the tricuspid valves (compare with Fig. 317).

the simultaneous events in the jugular pulse, the apex beat, the carotid, and the radial pulse. The perpendicular lines facilitate the comparison of the tracings at definite points in the cycle, and have the same significance in the later tracings: 1, the beginning of the auricular systole; 2, the beginning of the ventricular systole; 3, the opening of the semilunar valves and the appearance of the carotid pulse; 4, the beginning of the radial pulse; 5, the

closing of the semilunar valves; and 6, the opening of the tricuspid valves. The time is recorded in fifths of seconds in this and other tracings.

Although the carotid pulse and the apex beat can sometimes be usefully employed as a standard, it will be found that the radial pulse is, on the whole, the most convenient in practice. A certain loss of time takes place in the transmission of the wave to the radial pulse, and this can be estimated by taking simultaneously with the radial a few beats of the carotid, as in Figs. 318 and 321, in which the space between 3 and 4 shows the loss of time between the appearance of the carotid and the radial pulse. This loss being allowed for, one can always find a definite period in a jugular tracing which corresponds to any event occurring in the neck due to the ventricular systole. I also employ a very useful period, namely, that portion of the ventricular systole during which the semilunar valves are open (period marked *E* in some tracings). Its

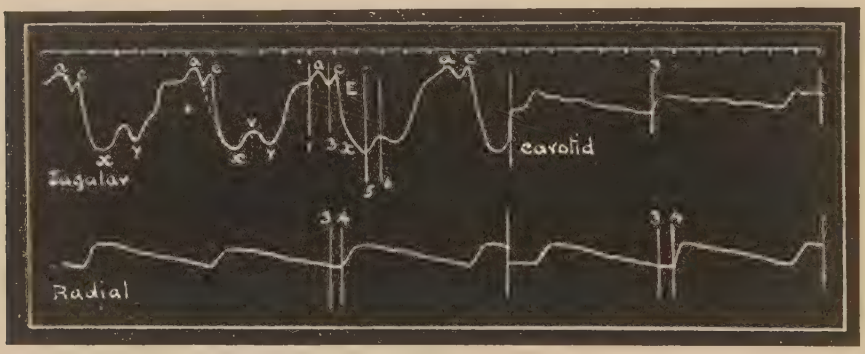


Fig. 321. Simultaneous tracings of the jugular and the radial pulses in the first part of the tracing and of the carotid and the radial in the latter part. The jugular pulse is of the auricular type. *a*, auricular wave; *c*, the carotid wave; *v*, the ventricular wave; *x*, the auricular depression; *y*, the ventricular depression. These letters have the same significance in all the other tracings, and the numbered perpendicular lines have the same significance as those in Fig. 320.

duration can be found in the radial tracing, extending from the beginning of the upstroke to near the bottom of the dicrotic notch. It corresponds to the time between the perpendiculars 3 and 5 in all the carotid, jugular, and apex tracings given here. In the neck the period *E* begins with the carotid pulse. There is a slight delay between the opening of the aortic valves and the carotid pulse, but it is so short (one-fiftieth of a second) that it may be ignored. The space *E* in the radial tracing begins about one-tenth of a second behind the same period in the neck.

Another important standard is that of the opening of the tricuspid valves (perpendicular line 6 in all the tracings), which in jugular tracings is always indicated by the beginning of the fall of the wave *v*. In the apex tracing this event occurs at the bottom of the fall, after the systolic plateau, as in Figs. 297 and 320, and in the radial, at the bottom of the dicrotic notch.

The carotid wave.—In the tracing in Figs. 320, 321, 322, in addition to the waves *a* and *v*, which have already been described, there is another wave marked *c*. In Fig. 316 it will be seen that the subclavian and carotid arteries lie in such close proximity to the jugular vein that the receiver covers a portion of these arteries. In consequence of this, the stroke of the arterial pulse affects the tracing from the jugular, and produces the wave *c*, which I have called the carotid wave. A considerable amount of discussion has taken place in regard to the cause of *c*; but if an observer will carefully take tracings higher and higher up in the neck, he can easily satisfy himself of its nature, for it gradually assumes the character of a tracing from the carotid artery like that in Fig. 327. The recognition of *c* as due to the carotid (or carotid and subclavian) helps much in the analysis of tracings from the neck, particularly when there is a delay between the auricular and ventricular systole, the duration of the *a*-*c* interval being the best measure of the delay.

Experimental observations show that faint waves may occur in the veins

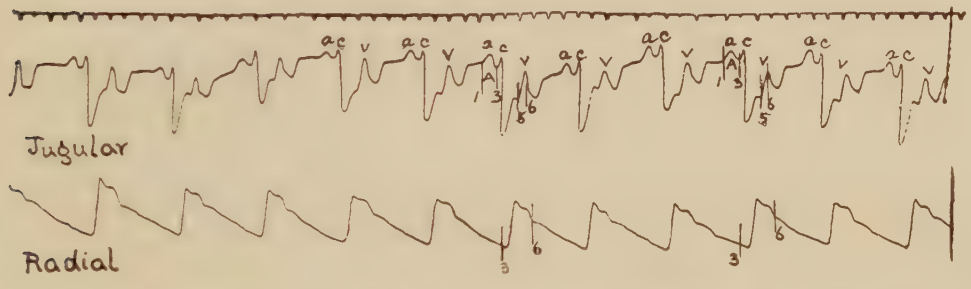


FIG. 322. Simultaneous tracings of the jugular and radial pulses from a healthy man. The letters and numbers have the same significance as in Fig. 321.

about the time of the carotid wave, produced in some obscure way by the systole of the ventricle, but the carotid and subclavian impact is the main, and for practical purposes the only one, that need be considered.

The true venous curve would follow the dotted line in Fig. 321.

The notch on the ventricular wave.—In a great many cases the wave *v* has a notch, followed sometimes by a considerable rise, on it just before its termination (perpendicular line 5 in Fig. 320). It corresponds in time to the closure of the semilunar valves, and the following rise in *v* occurs between the closure of the semilunar valves and the opening of the tricuspid valves (post-sphygmic interval, *F*, Fig. 317). The exact cause of this notch is still a matter of dispute—no satisfactory explanation being yet forthcoming. I have often employed it as a useful guide in measuring the period when the semilunar valves close, and it is represented by the perpendicular line 5 in all the tracings.

The diastolic wave.—Occasionally a wave may be detected in slow-acting hearts, shortly after the opening of the tricuspid valves (*h*, Fig. 323). A. G. Gibson and Hirschfelder describe this as due to the inrush of blood into the

ventricle, floating up the cusps, and causing a transient closure of the tricuspid valves. Thayer and Gibson also describe a sound heard occasionally at this time.

Changes due to variation in the rate of the heart.—When the heart's rate increases, the shortening of the cycle takes place mainly at the expense of the

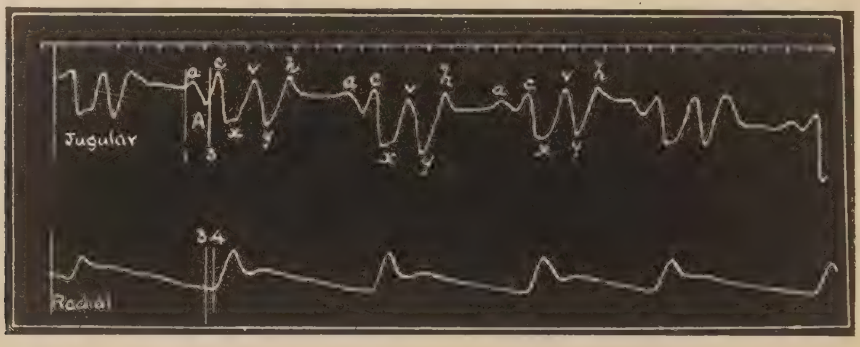


Fig. 323. Shows a diastolic wave (*h*) in the jugular tracing. (Rate 48.)

diastolic period. In the venous tracing, the first effect is shown by the disappearance of the period of stasis, the wave *a* following immediately on the ventricular wave *v* (Fig. 324); with still greater increase in the rate, *v* and *a* become blended (Fig. 325). In Fig. 326 the variation in the jugular pulse due to variation in rate is seen. Here there is an irregular rhythm with periods of slow rate and periods of increased rate. It will be seen that the

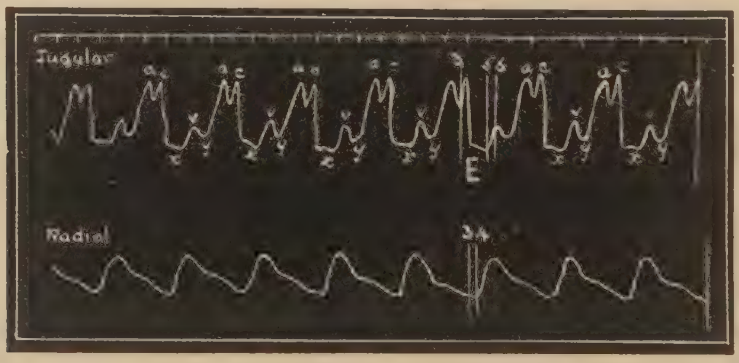


Fig. 324. In increase of the heart's rate the period between *y* and *a* becomes shortened, so that the auricular wave (*a*) follows immediately after *v* (compare with Figs. 322 and 323).

fall *y* disappears during the rapid period, while the waves *a* and *v* become blended.

Method of analysing a tracing.—In the tracings given so far, the waves have been distinct and well marked. It often happens that the jugular pulse is extremely small, so that we only get a slight movement due

to the auricle, the main portion of the tracing being due to the carotid, as in Fig. 327. At other times the movements of the neck seem mere dancing vibrations, and the record obtainable shows a series of small undulations. But with the radial pulse as a standard, one can definitely assign each

Fig. 325. With great increase in the rate the waves *v* and *a* become blended.

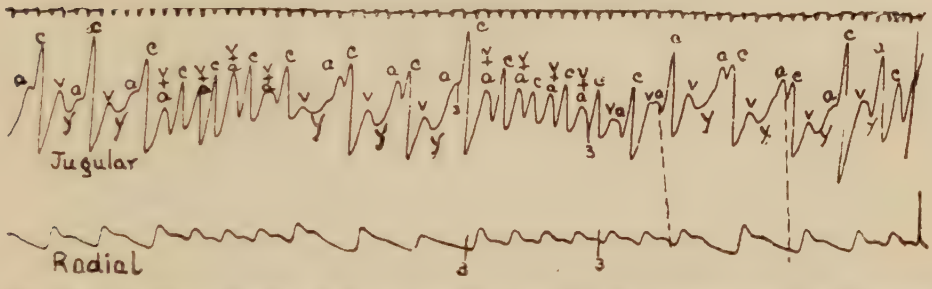


Fig. 326. Shows an irregular pulse of the youthful type. During the period of the infrequent rate the *a* and *v* waves in the jugular pulse are separated. During the more rapid rate the shortening of the diastolic periods causes the *a* and *v* waves to fall together.

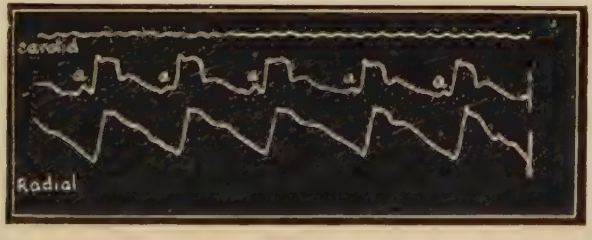


Fig. 327. In tracings from the neck the small wave (*a*) due to the auricular systole may be the only evidence of the jugular pulse.

undulation to the force producing it. Thus, in Fig. 328, we can analyse the tracing by the following procedure: Make a downstroke (4) parallel with the perpendicular line at the beginning of the radial tracing, at the beginning of a radial pulse-beat. As the carotid pulse occurs nearly one-tenth of a second before the radial, draw the perpendicular line 3 one-tenth of a second in

front of 4. Measure the distance from the perpendicular line at the beginning of the tracing to 3. Draw a downstroke in the jugular tracing, at the same distance from the one at the beginning. This will be found to fall at the beginning of a small wave, which, therefore, must have been due to the carotid, and so mark it *c*. The auricular wave occurs one-fifth of a second in front of *c*, and so the wave *a* can only be due to the auricular systole. In Fig. 320, p. 467, it was shown that the opening of the tricuspid valves (perpendicular line 6) often coincides with the bottom of the dicrotic notch in the radial tracing. If now a perpendicular line (6) be drawn at this period in the jugular tracing, it will be found to fall at the end of a wave, *v*, which must therefore be the ventricular wave.

By strictly following such a method as the foregoing, little difficulty will be experienced in analysing the great majority of tracings.

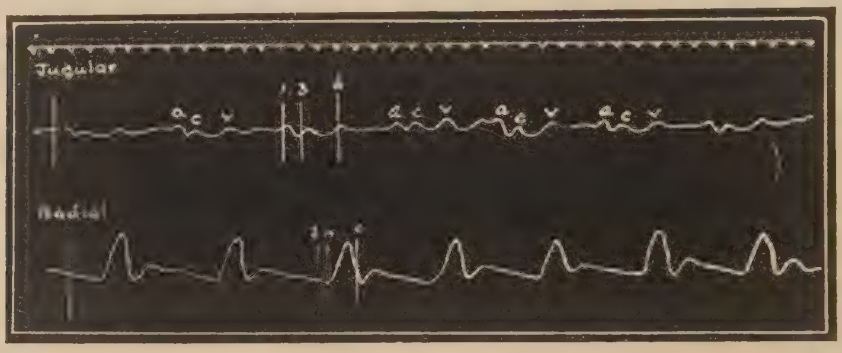


FIG. 328. The movements in the neck seemed dancing vibrations, but an analysis of a tracing refers each movement to a definite cause.

The ventricular form of the venous pulse.—In Figs. 329 and 330 are tracings of the jugular pulse. At a glance these are recognized to be totally different from the form of the venous pulse just described. The waves, *v*, in Figs. 329 and 330 are due to the blood being forced back through the tricuspid orifice into the veins by the contraction of the right ventricle. In a sense its origin is identical with the wave *v* in the auricular venous pulse, as in Figs. 318 and 319, but appears earlier in the cardiac cycle (synchronous with the carotid pulse) because *there is not now a dilating auricle interposed between the vein and ventricle.* When we come to analyse tracings of the ventricular venous pulse with a standard movement, as in Fig. 329, we discover there is no evidence of an auricular wave, nor of a fall corresponding to the fall *x* in the auricular venous pulse—in other words, there is one great wave (*v*) synchronous with the ventricular systole, and one great fall (*y*) synchronous with the ventricular diastole. Another point to be noticed is that the rhythm frequently is irregular, and that when there is a long diastolic period there is a rise in the tracing due to the vein filling, as after the long pauses in Fig. 330.

In the ventricular form of the venous pulse a wave occurs which I usually mark *c'*, of whose origin I am not certain. It occurs at the same time in the carotid, but it may be produced by a back flow into the veins at the beginning of ventricular systole (see Figs. 110, 111, 115, 331). The whole subject of the ventricular form of the venous pulse would well repay re-investigation.

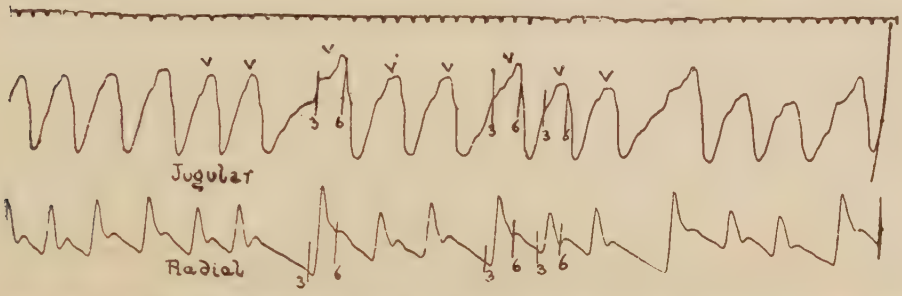


FIG. 329. Simultaneous tracings of the jugular and radial pulses, showing one large wave (*v*), synchronous with and due to the ventricular systole, and one large fall (*y*) synchronous with and due to the ventricular diastole. There is no sign of an auricular wave, and the jugular pulse is therefore of the ventricular type, and the rhythm of the heart is continuously irregular.

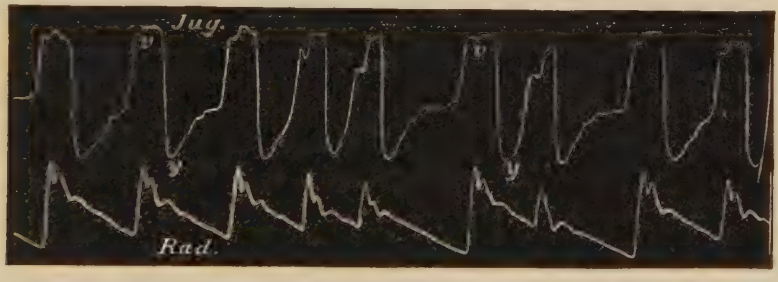


FIG. 330. Simultaneous tracings of the jugular and radial pulses, showing the ventricular jugular pulse, *i.e.* one large wave (*v*) synchronous with and due to the ventricular systole, and one large fall (*y*) synchronous with and due to the ventricular diastole. The rhythm of the heart is continuously irregular. When there are long pauses the tracing of the jugular gradually rises before the next large wave, on account of stasis in the veins. When the pause is short there is no sign of stasis, or only a slight wave, as on the second beat, where it might simulate a wave due to the auricle, but the real cause is seen to be stasis in the veins when the pause is longer.

There are three conditions which produce the ventricular form of the venous pulse :—

(1) The onset of auricular fibrillation. This is by far the most frequent cause of the change in the venous pulse from the auricular to the ventricular type. In the great majority of cases it is accompanied by the complete and disorderly irregularity of the heart's action, as shown in Figs. 329 and 330. The appearance of the ventricular form of venous pulse in auricular fibrillation is always accompanied by a series of other manifestations, which were

described in the chapter on auricular fibrillation (Chapter XXVIII.). Occasionally the rhythm is quite regular, but then the rate is always slow, as in Fig. 331.

(2) Great distension of the right auricle. Here there is great engorgement of the right heart, so that the auricle becomes embarrassed in its action, and the *a* wave in the jugular pulse diminishes in size and disappears, while the *v* wave increases in size and occupies the whole period of ventricular systole. With recovery and diminution of the engorgement, the auricular wave reappears. It is to be noted that the rhythm in these cases is always regular. This manner of production of the ventricular venous pulse is, however, very infrequent, far less common than my earlier observations led me to suppose, for the vast majority of cases of the ventricular venous pulse are due to auricular fibrillation.

(3) In certain forms of regular tachycardia, where the heart's contraction starts from an abnormal part of the genetic system. In these cases the engorgement of the heart is so great that each ventricular systole sends back

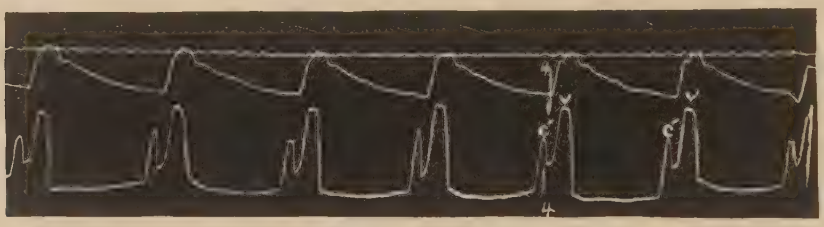


Fig. 331. Jugular and radial tracings from a case of auricular fibrillation. The jugular pulse is of the ventricular form, and the rhythm is quite regular. The slow rate and regular rhythm are the result of digitalis. Note the well-marked *c'* wave.

through the auricle into the veins large waves of blood. So forcible may these waves be, that they are mistaken sometimes for beating of the carotid (compare Figs. 332 and 333). Such waves may be present when the auricle and ventricle contract together as in nodal tachycardia (Figs. 71 and 72), or they may occur in auricular flutter (Figs. 139, 148, 165).

Anomalous forms of the venous pulse.—It is not always easy to appreciate the various events in the jugular pulse during abnormal action of the heart. In certain cases of auricular fibrillation this difficulty is not infrequently met with. The tracing from the neck in such instances as in Fig. 64 shows marked variation in the character of the waves. These variations are due sometimes to the differences in the fullness of the veins, produced by variation in the rate and the phases of respiration. Another class often causes so much difficulty that I failed to interpret the waves. In auricular flutter and paroxysmal tachycardia the varying relationships of the action of the auricles and ventricles produce such a medley of waves that frequently I have failed to interpret satisfactorily the tracings obtained from the neck.

Conditions giving rise to a venous pulse.—To a great extent we are

even at this day quite at a loss to explain all the conditions that give rise to a venous pulse. Most people in good health show it, while in cases of marked heart failure it may be entirely absent. Some people when in robust health show no signs of it, but if they become slightly debilitated

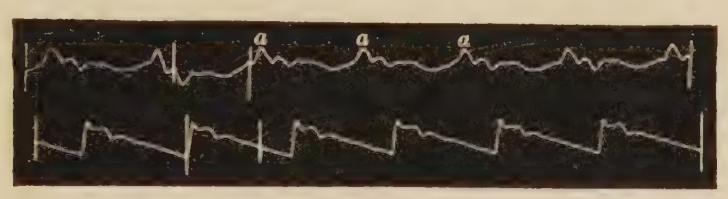


FIG. 332. Tracings of a slight movement in the jugular vein taken at the same time as the radial pulse. The wave *a* is due to the systole of the right auricle.

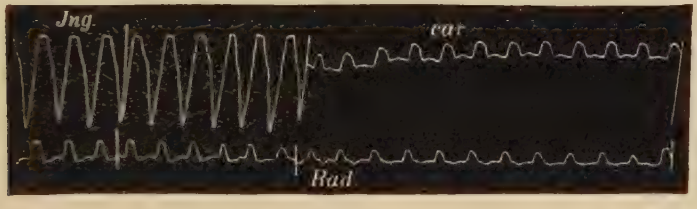


FIG. 333. Simultaneous tracings of the pulsation in the jugular bulb and in the radial, and of the carotid and radial pulses, during an attack of paroxysmal tachycardia, taken eighteen hours from the beginning of the attack.

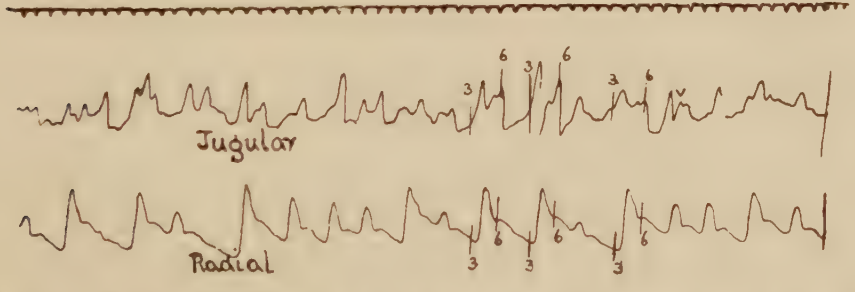


FIG. 334. Shows the variability of the jugular pulse of the ventricular form due to the respiration and variation in rate. Taken from a patient with auricular fibrillation.

the venous pulse may appear in the neck. It is frequently absent in the aged, though they are otherwise well. In some cases of pernicious anæmia the venous pulse may be a very prominent symptom ; in others it may never appear. Some women during pregnancy develop a large venous pulse, others only develop it during the puerperium, while others never show the slightest sign of it. In irregular action of the heart it may assume in certain cases enormous proportions ; in other cases there is not the slightest sign. A man

may show it during one attack of heart failure, and during another and more grievous attack it may be absent.

I have endeavoured to find out the reason for this variability, but though in some cases I have been able to connect its appearance and disappearance with definite changes in the heart, yet on the whole the matter is one which still puzzles me.

CHAPTER LXIX

Enlargement and Pulsation of the Liver

The relation of enlarged liver to heart failure.
Reflex symptoms.
Signs of enlargement of the liver.
Pulsation of the liver.
Conditions producing enlargement and pulsation of the liver.
Jaundice.
Different forms of the liver movement.
Differential diagnosis of liver enlargement.
Prognosis.
Treatment.

The relation of enlarged liver to heart failure.—In dealing with heart failure it was pointed out that the signs of heart failure were not to be found in the study of the heart itself, but in the other organs of the body, where the impaired blood supply due to the heart's inefficiency caused changes in the size or function of the organ. The enlarged liver is one of the most characteristic signs thus produced.

The symptoms arising from enlargement of the liver due to heart failure receive little consideration from clinicians, and are very frequently overlooked or misunderstood. To a certain extent, this is due to the fact that this enlargement may appear at such an advanced stage of cardiac failure that the diagnosis and treatment can be determined without paying particular attention to the liver symptoms. Graham Steell includes the enlargement of the liver as one of the cardinal symptoms of heart failure, and Salaman has given a very suggestive analysis of the pathological changes and the conditions inducing them; but clinicians generally have dealt with the subject in a most perfunctory manner. While it is true that the conditions producing these liver troubles imply an advanced stage of heart failure, yet the recognition of the symptoms has an important bearing on diagnosis and treatment in many cases. The symptoms due to changes in this organ are not always easy to understand, but that is no reason for ignoring them.

Reflex symptoms.—Usually in the early stages of liver enlargement, while the liver may be only one or two inches below the ribs, the muscle wall of the upper part of the right half of the abdomen becomes hard

and tender. This tenderness is invariably put down to the liver itself, and the manner in which this 'tenderness' is usually demonstrated is by eliciting pain on pressing the finger into the patient's abdomen. But if the extent of the hyperalgesia and the size of the liver be mapped out, it will be found that the former is far more extensive than the latter, and sometimes extends round to and affects the erector spinæ muscles. In some cases the skin and subcutaneous tissue also become tender, but they rarely become so sensitive as the muscles. Sometimes there seems an increased tenderness where the liver is reached, but this will be found to be due to the more effective compression of the muscle between the finger and the liver. There are many other ways by which the resourceful observer can demonstrate the tissue in which the tenderness is present.

The consequence of this muscular hyperalgesia is manifested in various ways. If the patient is going about, he may suffer severe pain across the upper part of the abdomen or in the back, this probably being due either to increased engorgement of the liver, or to the increase of the pain in consequence of the exercise of the hyperalgesic muscles. This tenderness and rigidity of the abdominal muscles interferes with the respiration of the patient. He cannot breathe deeply, and attempts to do so are painful, hence there results rapid and shallow breathing, with further embarrassment of the right heart and a tendency to pulmonary stasis.

With long-continued persistence of the enlargement, all the sensory phenomena disappear, the abdominal wall becomes lax, and sometimes the edge of the liver can be grasped.

Signs of enlargement of the liver.—It is not always easy to make out the enlargement of this organ. The contracted muscles often prevent the palpation, and even percussion helps but little. Ascitic and gaseous distension add further to the difficulty. But with care and gentleness in palpation, one may overcome the resisting muscles. Even when the edge of the liver cannot be made out, the peculiar sense of resistance conveyed to the exploring hand may reveal the enlarged liver. Other methods may be adopted, such as pushing the liver forward with one hand while the other explores the front. When the muscles relax and the hyperalgesia disappears, there is no difficulty in finding out the enlarged liver, except when there is great distension of the abdomen.

Pulsation of the liver.—I am disposed to think that the more common enlargement of the liver is due to stagnation of the blood from a diminished *vis a tergo*. There is no doubt that there is at times an additional factor in the throwing back of blood from the right heart, for we occasionally find pulsation of the liver. If the abdominal muscles are lax, there is no difficulty in recognizing this. If one hand presses on the liver behind and the other is laid on it in front, the latter is heaved up and down with the pulse. Even where there is a considerable amount of muscular contraction, exploring the edge of the liver with the liver receiver will often reveal the pulsation (pp. 422 and 423).

There are two forms of liver pulse, corresponding to the two forms of venous pulse — an auricular (Fig. 335) and a ventricular (Fig. 336). When a liver pulse and a jugular pulse are both present in the same individual, they are always of the same form. In Fig. 337 a tracing of an auricular jugular pulse is taken simultaneously with a tracing of the auricular liver pulse. In

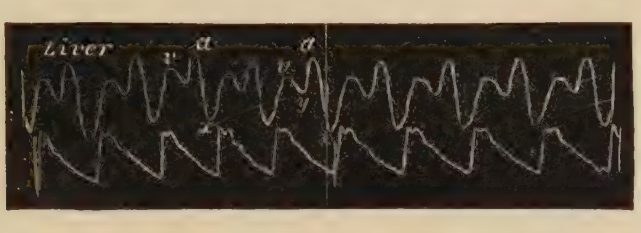


Fig. 335. The liver pulse is of the auricular form and shows a well-marked auricular wave *a*.

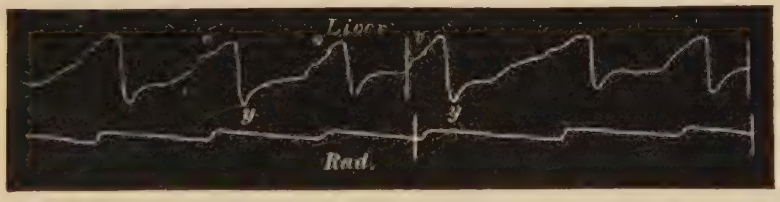


Fig. 336. The liver pulse is of the ventricular form, showing no auricular wave.

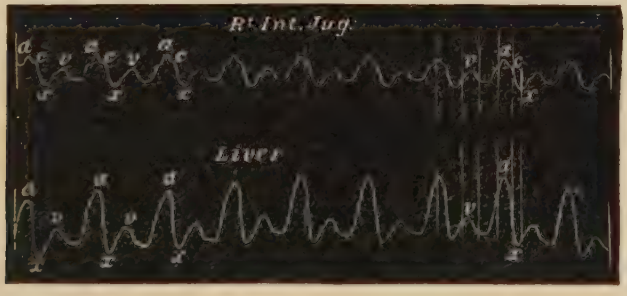


Fig. 337. Simultaneous tracings of the jugular and liver pulses, showing the correspondence between the waves *a* and *v*, and the absence of the carotid wave *c* from the liver pulse.

Fig. 338 the tracing of the ventricular jugular pulse is seen to be identical with the liver pulse in Fig. 336, both tracings being from the same patient.

Conditions producing enlargement and pulsation of the liver. — I have already remarked on the variety of conditions producing the jugular pulse; and there is a like difficulty in understanding the conditions which give rise to liver enlargement. Cases otherwise identical in their symptoms, and suffering from heart failure due to the same cause, may differ in this particular, some showing enlargement of the liver, and others failing to do so. Similarly, it

is not always quite clear why some pulsate while others do not. To a certain extent I think this is due to the condition of the right auricle. It takes some force to distend the liver, and normally the right auricle has not sufficient strength, so that as long as the right auricle contracts and dilates in its normal place in the cardiac cycle, it prevents the ventricle exercising its force on the liver. When, however, the auricle is not contracting in a normal

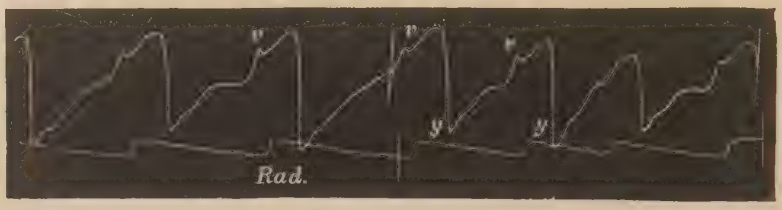


Fig. 338. Simultaneous tracings of the jugular and radial pulses. The jugular pulse is of the ventricular form and is identical with the liver pulse in Fig. 339, both being taken from the same patient.

manner, the ventricle drives the blood through the incompetent tricuspid orifice with such force that the liver pulsates. It is in the cases of auricular fibrillation that we most frequently find the liver pulsating. When the auricular fibrillation or certain forms of tachycardia are transient, the liver may quickly enlarge and pulsate, and with cessation of the attack as quickly subside and cease to beat—the liver pulse being of the ventricular form (Fig. 339).

Reasoning that it requires some force greater than the normal strength of the right auricle to produce pulsation of the liver, I at first drew the conclusion that an auricular liver pulse indicates hypertrophy of the right

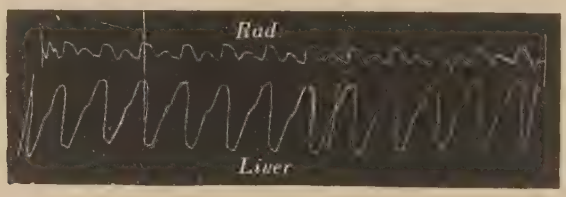


Fig. 339. Simultaneous tracings of the radial and liver pulses during an attack of paroxysmal tachycardia towards the end of life.

auricle, and as this occurs most characteristically in cases of tricuspid stenosis, I regarded the auricular liver pulse as diagnostic of tricuspid stenosis. All the cases that had shown this auricular liver pulse during life, whose hearts I examined post mortem, showed tricuspid stenosis. But I have now had a number of cases showing this form of liver pulse, in which I doubt if I am justified in assuming tricuspid stenosis. Turnbull and Wiel have recorded a case from my clinic in which there was an auricular liver pulse and no tricuspid stenosis.

Jaundice.—Jaundice is a frequent accompaniment of enlargement of the liver; though of itself of little importance, it should be kept in mind that a slight jaundice may be misleading. Many patients with advanced heart failure and with auricular fibrillation get rapidly thinner. In these the liver is sometimes greatly enlarged, so that the wasting of the patient, the enlargement of the liver, and the jaundiced tint present the features of malignant disease of the liver; and I have seen cases presenting these symptoms thus wrongly diagnosed.

Different forms of the liver movement.—Many writers refer to an 'arterial pulsation' of the liver, and I have often wondered what they mean, particularly as no details are ever given. I have never found any condition that could come under this heading, and I suspect that some observers have mistaken the movement of the liver, when it is pulled up and down with the systole and diastole of the ventricle, for a pulsation, particularly as it is sometimes mentioned in connexion with aortic regurgitation. Wherever there is much cardiac enlargement, movement of the liver is produced. Even in

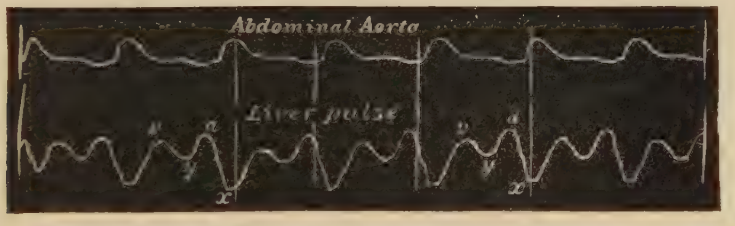


Fig. 340. Tracings from the abdominal aorta and the liver.
The liver pulse is of the auricular type.

patients with normal hearts but with lax abdominal wall, this up-and-down movement can be recognized. It has already been referred to on p. 439. Here I need only point out that the conditions in which it occurs are generally very different from those of the heart failure in which the true liver pulsation occurs. A tracing of this movement sets the matter at rest, as the fall due to the ventricular systole does not begin until the opening of the aortic valves and the expulsion of blood from the chest, which is practically synchronous with the carotid pulse. In the ventricular form of liver pulse, on the other hand, there is a rise during ventricular systole. A tracing of the liver movement can be distinguished from the auricular liver pulse, in that the fall in the latter case precedes the carotid pulse.

There is generally little difficulty in distinguishing the pulsation of the abdominal aorta from that of the liver pulse. In cases where the ventricular liver pulse is present, it is very rarely that the abdominal aorta can be felt, and the characters of the tracings are not likely to be confused. The auricular form of liver pulse is still more distinct in character from that of the abdominal aorta, as shown in Fig. 340.

Differential diagnosis of liver enlargement.—There are cases occasionally

met with where there are dropsy, ascites, enlargement of the liver, which resemble heart failure so much that they are not infrequently mistaken for it. It may be taken for granted that when there is liver enlargement without dilatation of the heart, the enlargement is not due to heart failure. In a few cases which I have seen, my suspicion has been aroused by the absence of dilatation of the heart and absence of cardiac dyspnœa. Careful inquiry into the habits of the individual may reveal a history of long-continued indulgence in alcohol. With such a history, one can conclude that the case is one of hypertrophic cirrhosis of the liver. Moreover, these livers never pulsate.

Prognosis.—Liver enlargement and pulsation from cardiac disease indicate a very advanced stage of heart failure. In cases where the heart failure is secondary to a rheumatic affection of the heart with mitral disease, it may only appear during the attacks of heart failure to which the patients are liable.

Fig. 341. Simultaneous tracings of the apex beat and of the liver pulse. The liver edge was below the level of the umbilicus, and the pulsation of great size.

The enlargement subsides with improvement ; and if the heart's restoration be good, the patient may have no signs of the liver enlargement for many years. Some patients with auricular fibrillation secondary to rheumatic affection of the heart may for years have a big pulsating liver (Fig. 341) ; in others the enlargement appears during temporary heart failure. In some of these the exhibition of digitalis may cause the subsidence of the liver in a couple of days. Fig. 342 was taken from the liver of a man which was pulsating as low as his umbilicus. In a couple of days all signs of the liver below the ribs had gone. The enlarged liver coming on in paroxysmal tachycardia is a serious symptom, as it indicates a tendency to dilatation and inefficiency of the heart. Also in the heart failure due to cardio-sclerosis with permanent auricular fibrillation, it is a very grave symptom, especially when the heart failure is due to extensive degeneration of the cardiac muscle, and does not respond to the influence of rest and digitalis. Enlargement of the liver in muscle failure, due to chronic alcoholism, is a bad sign.

Treatment.—Though no treatment is directed specially to the liver enlargement, the effect of the rigid tender muscles in embarrassing the respiration should be borne in mind, and suitable attempts made to put the patient

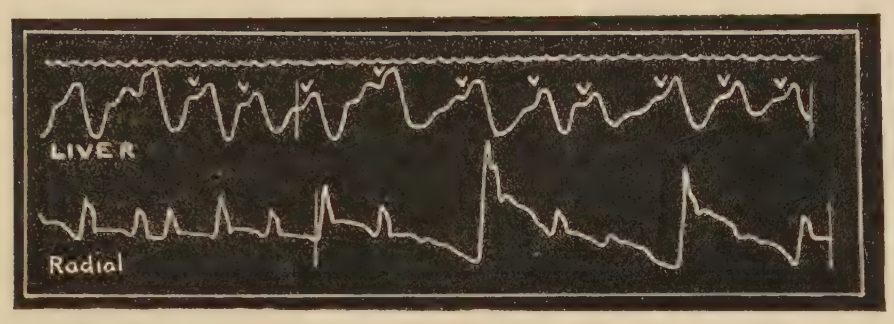


Fig. 342. Tracings of the liver and radial pulses during a period of extreme heart failure (auricular fibrillation).

in a comfortable position, so that he can breathe more easily ; and the pain due to the enlarged liver should call for the cessation of all exertion that induces it. A smart mercurial purge may sometimes afford considerable relief.

INDEX OF NAMES

Adami, 451
Allbutt, Sir Clifford, 457

Barenne, 328
Bowditch, 103
Brauer, 379
Broadbent, 378, 447
Buchanan, Miss, 304

Cooper, 378
Cullis and Dixon, 264
Cushny and Edmunds, 160
Cushny and Matthews, 130
Cushny and Wenckebach, 130

Da Costa, 352
Dale and Kellaway, 79
Delépine, 438
Dixon, 264
Dudgeon, 190

Edmunds, 160
Einthoven, 424
Engelmann, 8, 285

Foster, 373
Friedreich, 378

Gairdner, Sir William, 15, 363
Gaskell, W. H., 8, 66, 98, 100, 131, 188, 310
Gibson, A. G., and Hirschfelder, 469
Gibson, G., 374, 381
Griffith, Wardrop, 288
Grossi, L., 95
Gull, Sir William, 212

Hawthorne and Da Costa, 352
Herring, Prof. P. T., 64
Hill, L., 455
Hirschfelder, 469
Horder, Sir Thomas, 399
Hunter, John, 370, 438

Ishihari and Nomura, 101

Jolly and Ritchie, 215

Keith, Sir Arthur, 92, 235, 309
Kellaway, 79
Keyt, 443
Krogh, 78, 79

Laennec, 15
Langley and Walker, 234
Laslett, E. E., 121
Lewis, Sir Thomas D., 160, 164, 235, 373
Lister, Lord, 446

MacWilliam, 185, 215, 304, 305, 326, 454
Marey, 7, 130, 190, 446
Marris, 416
Matthews, 130
Mayer, 63
Mayo, 370
Mosso and Delépine, 438

Nicholson, 378
Nomura, 101

Oliver, 455
Orr, James, 246

Parkinson, John, 264
Phear and Parkinson, 264
Poiseuille, 446

Ramsay, Maitland, 82
Ritchie, W. T., 215
Romanes, 63, 98
Rothberger, 156, 160
Rowland, 455
Roy and Adami, 451
Russell, 455

Salaman, 477
Sanders, Gordon, 285
Smith, Prof. Lorrain, 81
Starling, Prof. E. H., 96
Steell, Graham, 369, 373, 477
Sutherland, G. A., 312
Sutton, H. G., 212

Tallant and Cooper, 378
Tawara, S., 98, 100, 131, 250
Thayer and Gibson, 470
Tufnell, 388
Turnbull and Wiel, 480

Von Jacquet, 190

Walker, 234
Waller, 424
Waterston, Prof. D., 64, 429
Wenckebach, 130, 285, 379
Wiel, 480
Wilson, R. M., 352
Winterberg, 160
Withering, 158

GENERAL INDEX

Abdominal aorta, tracing from, 481
  pulse of, compared with liver pulse, 481
Abdominal belt, value of, in relieving faintness, 416
Abnormal rhythms, table and diagram of, 106
a-c interval, in cases of extra-systole, 131
  in partial heart-block, 245
  increased, 131, 256, 258
  variations in, 252
Activity, disorderly, in nervous diseases, 76
Adhesive mediastino-pericarditis, 377. *See* Mediastino-pericarditis
Adrenalin in heart-block, 264
Afferent nerves of heart, 93
Age, advancing, effects of, 334, 335
Age changes in arteries as affecting blood-pressure, 456, 457, 458
Alcohol, effect of, on muscle of heart, 310
  in angina, 344
'All or nothing', law of, in relation to heart, 31, 102, 103
Amyl nitrite, in angina, 327, 344, 345
Anacrotic pulse in aortic stenosis, 372
Anaphylaxis, 70
Anatomy of heart, 88, 90 *et seq.*
Aneurysm of the aorta, 382-388, *and see under* Aorta
Aneurysm, arteries chiefly affected by, 383
  causes of, 383
  definition of, 382
  dissecting, 383
  pulse in, 449
  rupture of, 384, 386, 387
  sacculated, 383
  varicose, 383
  varieties of, 383
Aneurysmal varix, 383
Aneurysms, miliary, 383
Angina pectoris, 324-350
  alcohol in, 344
  amyl nitrite in, 327, 344
  and auricular fibrillation, 198
  and the principle of the reflex arc, 332, 333
  associated sensations in, 337
  attacks of, 336, 337
  blood-pressure in relation to, 459
  bodily effort inducing, 343, 344
  chloroform in, 344
  cold causing, 344
  compression or constriction of chest in, 337
  conditions causing attacks of, 334-336
Angina pectòris (*contd.*)—
  coronary arteries in, 327, 335, 341
  death in, 338, 339, 340, 341
  depression or oppression in, 337
  gaseous eructation in, 338
  hyperalgesia with, 325, 328
  in aortic valvular disease, 375
  in mitral stenosis, 368
  increased flow of saliva in, 338
  increased flow of urine in, 338
  meals in, 344
  morphine in, 345
  nitroglycerine in, 344, 345
  oncoming of, 334-336
  perspiration after, 338
  primary, 334-339
  principles of treatment in, 407
  prognosis in, 340-342
  pulse in, 327
  secondary, 348-350
    bromides in, 350
    clinical features of, 348
    nitrites in, 350
    prognosis of, 349, 350
    significance of, 349
    treatment of, 350
  sense of impending death in, 337
  surgical treatment of, 345-347
  treatment of, 342-347
  two kinds of, 329
  vaso-motor, 336
  vaso-motor disturbances in, 337, 338
  ventricular fibrillation in, 339
  vomiting in, 338
Anginal attacks, prophylactic treatment of, 344, 345
  treatment of, 344, 345
Antiarin, action of, on muscular tone of heart, 310
Aorta, aneurysm of, 382-388
    prognosis in, 386, 387
    treatment of, 387, 388
  abdominal, aneurysm of, symptoms of, 386
    pulsation of, distinguished from liver pulsation, 481
  ascending, aneurysm of, symptoms of, 384
  descending, aneurysm of, symptoms of, 385, 386
  dilatation of, 388, 389
    prognosis of, 389
    symptoms of, 389
    treatment of, 389

Aorta (*contd.*)—
double, 383
thoracic, aneurysm of, symptoms of, 383-386
transverse, aneurysm of, symptoms of, 384, 385
Aortic knob, the, 384
Aortic notch in sphygmogram, 451, 452
Aortic regurgitation, 359, 373-376
auricular fibrillation with, 375
capillary pulse in, 374
dilatation of aorta in, 388
heart failure in, 375
murmur of, 373
pulse in, 374
susceptibility to nerve stimulation in, 375
visible arterial pulsation in, 447
Aortic stenosis, 372, 373
murmur with, 373
pulse in, 372
Aortic valves, rupture of, 372
Aortic valvular disease, 372-376
ætiology of, 372
and heart-block, 259
symptoms and course of, 375
Apex beat, 433, 434
alteration of, from retraction of lung, 443, 444
auricular wave in, 437
due to left ventricle, 434-441
due to left ventricular systole, tracing of, 434-437
due to right ventricle, 441-443
effect of indrawing of tissues on, 438, 439
in aortic regurgitation, 374, 375
in extra-systole, 142
normal situation of, 433
real and palpable, 433
systolic plateau in the, 436
tracing of the, 434
variations in position of, 433
Apoplexy, cerebral, and arterial degeneration, 458
Arborization block, 258
Arcus senilis, 80
Arm, pain in, in angina, 326
Arryhthmia, sinus, 20, *and see* Irregularity
Arsenic, effect of, on heart muscle, 310, 312
Arsenical poisoning, cardiac dilatation in, 312
Arterial degeneration, blood-pressure in relation to, 456, 457, 458
results of, 403, 404
tortuous superficial vessels in, 447
Arterial disease and heart-block, 259
Arterial pressure, 453-460
capillary obliteration in relation to, 460
cause of, 453
heart-failure in relation to, 460
high, ignorance of its causes and consequences, 458
high, nitrites in, 460
high, prognosis of, 458
high, treatment of, 459, 460
in angina pectoris, 459
variations in, 459
and see Blood-pressure
Arterial pulsation, visible, 447
Arterial walls, examination of, 448
Arteries, atheroma of, 448
calcareous degeneration of, 448
degenerative changes in, 335, 382, 383
digital examination of, 447-449
inspection of the, 447
tortuous, 446, 447
variation in size of, 448
Ascites, 34
'Aspiration, cardiac', 438
Asthma, cardiac, 34
Atheroma of arteries, 448, 456
Atropine, action of, on heart, 69
on vagus, 97, 406
effect of, in heart-block, 257
Augmentor nerve of heart, 95
Auricle and jugular vein, causes of variation of pressure in, 465, 466
Auricle, control of, by s.-a. node, 41
impulses discharged by, in auricular flutter, 235
paralysis of. *See* Auricular fibrillation
the *ultimum moriens*, 71
Auricular contraction, nature of, in auricular flutter, 234, 235
Auricular fibrillation, age incidence of, 183-185
and heart-block, 249, 250, 259, 290-293
angina pectoris and, 198
breathlessness in, 162
cases illustrative of, 159, 170-180, 201, 202
definition of, 160, 161
diagnosis of, 162-169
distinctive features of, 181
due to disorderly activity, 181
electro-cardiogram of, 168, 169, 192-194
exhaustion in, 414
facies in, 162
fibrillation waves in, 168
following auricular flutter, case illustrating, 246
graphic records of, 166-169
heart-sounds in, 163-166
historical account of, 158-160
in relation to heart failure, 162
onset of, producing ventricular form of venous pulse, 473, 474
origin of, 185, 186
paroxysmal attacks of, 179
principles of prognosis in, 199
the production of heart failure in, 196-198
pulse in, 163
pulse irregularities of, 190, 191
small beats in, 194
sudden death in, 201-203
symptoms of, 181
transient attacks of, 202
treatment of, 204-214
variable results of, 199, 200
with aortic regurgitation, gravity of, 375
without limitation of effort, 163
Auricular flutter, 215-220
and heart-block, 250, 259

Auricular flutter (*contd.*)—
cases illustrating, 221-233, 241
cases illustrating effect of digitalis in, 241, 246
cause of, 234
danger of digitalis in, 246
diagnosis of, 215, 216
electro-cardiogram of, 220
exhaustion in, 414
fibrillation following, 246
fibrillation following, case illustrating, 246
heart failure in, 238, 239
jugular pulse in, 216, 218
origin and nature of, 234-237
origin of paroxysmal attacks of, 236, 237
prognosis in, 239, 240
pulsus alternans and irregular pulse in, 218
radial pulse in, 216-218
recognition of, 215
respiratory symptoms in, 239
symptoms of, 216
treatment of, 241-248
Auricular form of venous pulse, 465, 466
Auricular wave in cardiogram, 437, 438
in cardiogram, interpretation of, 437, 438
in graphic records, 90, 437, 438, 465
in the jugular tracing, 465
Auriculo-ventricular bundle in relation to extra-systole, 133
in relation to ventricular beat, 139
independent activity of, 106
mode of contraction of, 101
refractory state of, 133
structure of, 91, 100, 101
Auriculo-ventricular node, activity of, 125-127
and the contraction of the ventricle, 191, 192
decreased excitability of, due to digitalis, 245
in disease, 406
in relation to heart movements, 60, 62, 106
in the tortoise heart, 188
independent activity of, 106, 187
the independent, imperfect control of the, 194, 195
relation of, to fibrillating auricle, 188
stimulation and inhibition of, 189, 190
structure of, 58, 90
the uncontrolled, 187-195
vagus and sympathetic in relation to, 96, 106
or bundle, its rôle in the extra-systole, 132, 133
Auriculo-ventricular valves, opening of, 437
Auscultation, value of, in diagnosis, 13
in prognosis, 15
Axon process of nerve cell, 57

Bellows murmur, 373, 374
Blood-pressure, methods of measuring, 453-456, 459
high, 456, 457
prognosis of, 458, 459
treatment of, 459, 460
and see Arterial pressure
Blood supply in relation to microbic infection, 81
of the heart, 91
Bradycardia, nodal, 291
Breathlessness, 33
in auricular fibrillation, 162, 163
in auricular flutter, 239
Broadbent's sign in adhesive mediastino-pericarditis, 377, 378
Bundle-block, 258

Canalis auricularis, 86
function of, 100
in relation to impulse production, 87
in the tortoise, 188
Capillaries as cause of failure of circulation, 79
as organs, 77
capacity of, 26
changes in, with advancing years, 79, 80, 335
contractility of, 78
control of, 78
effect of contracting muscle on, 78
in relation to cessation of heart-beat, 26
the most primitive of structures, 77, 81
nerves and the, 78
obliteration of, 79, 80, 81
Capillaries, rôle of, in production of symptoms, 77-83
uncontrolled, and failure of the circulation, 78, 79
Capillary activity, regulation of, 77
Capillary circulation when death is impending, 78, 79
Capillary field, cutaneous, fluctuation in, 64, 65
Capillary obliteration as cause of degenerative changes, 402, 403
as explanatory of heart troubles of advancing years, 403
cause of, 81
in the eye, 82, 83
Capillary reservoir, capacity of, 79
'Cardiac aspiration,' 438, 439
Cardiac asthma, 34
Cardiac chambers, contraction of, 91, 92
Cardiac contractions, character of, 89
Cardiac cycle, description of events in, 463, 464
standards for recognising events in, 431
Cardiac pain from coronary inadequacy, 80, 81
Cardiac revolution, events during, as revealed by sphygmogram, 450, 451
Cardiac tube, the primitive, 85, 86
Cardiogram, auricular wave in, 437, 438
from apex beat due to right ventricle, 441, 442
interpretation of the, 426, 427, 434-437
inverted, 438
significance of, 442, 443
ventricular systole as recorded in, 444
Cardiology, modern, review of, 5
Carotid wave in the jugular tracing, 469
Cell impulse and electricity, 71, 72
and its investigation, 49, 99
Cell, vital activities of the, 51, 52
Cells and impulses, 38, 39
of heart muscle, properties of, 60, 61
plant, functioning of, 52-54

'Centre', explanation of term, 68
'Centres', nerve, 66-69
Cerebral symptoms in heart-block, 262, 263
of heart failure, 34
Chemical agents, effect of, on vital activities, 59
Chest-wall in relation to recognition of heart movements, 431, 433
Cheyne-Stokes breathing, 33
as prognostic sign, 313
in myocardial affections, 312, 313
Chloroform in anginal attacks, 345
Circulation, how it is maintained, 84
'Circus rhythm', 185, 235
Classification, the sound basis for, 104
of symptoms, 73-76
basis of, 73, 74
object of, 73
of variations in rate and rhythm, 104-107
Clinical and laboratory methods of diagnosis, 12, 13, 19
Clinical records of auricular flutter, 221-233, 241, 246
of combined affections of genetic system, 297-303
of heart-block, 265-283
of the heart in 'debility', 351, 352, 353
of myocardial affections, 314-318
of pulsus alternans, 289
of secondary angina, 349
of sudden death in heart disease, 201, 202
of syphilitic heart affections, 320-323
of ventricular fibrillation, 281
Clinical observations on use of digitalis in auricular fibrillation, 209, 210
Clubbing of fingers in congenital heart affections, 380
Cold, effect of, in causing anginal pain, 336, 344
Conducting system, suspension of activity of, 304, *and see* Genetic system
Conduction, as seen in hair of sundew leaf, 53
by neurones, 77
from cell body to cell body, 77
Conductivity, 100, 101
Congenital affections of the heart, 380, 381
ætiology of, 380
prognosis of, 381
symptoms of, 380
treatment of, 381
Congenital defects of aortic valves, 372
Consciousness, loss of, 415, *and see* Fainting
with heart block, 262, 263
Constriction, sense of, in angina, 337
Contractile organs, 55
Contraction as shown in hair of sundew leaf, 53
of the cardiac chambers, 91, 92
of heart, impulses which stimulate, 102
of heart muscle cells, 426
of muscle, 55, 56, 85
Control, 49, 59-62
by the genetic system, 101, 102
function of, as shown in hair of sundew leaf, 54
in relation to genetic system, 101
Control (*contd.*)—
loss of, effects of, in heart, 61, 62
loss of, effects of, on sino-auricular node, 187
of heart's movements by nerves, 102
of vital activities, 59-62
primitive, by sinus venosus, 86, 87
Controlled and uncontrolled movements in medusa, 62, 63
Convulsions from cessation of output from heart, 415
in heart-block, 262
Cor bovinum, 372
Coronary arteries, 91
capillary obliteration and the, 80, 81
sclerosis of, 335
in old age, 403
Corrigan's pulse, 374
Cough, brassy, of aneurysm, 385
Cutaneous sensation, fluctuation in, 64
Cyanosis, 35
in congenital heart affections, 380
in heart failure, 35

Death, genetic system in the production of, 304, 305
the manner of, 24, 71
manner of, in angina pectoris, 338, 339, 340, 341
sudden, in angina pectoris, 339, 340
ventricular fibrillation a chief cause of, 304
'Debility', 351
the heart in, 351-353
Dendrons of nerve cell, 57
Denervated heart, the, 96
Depression, sense of, 412
Diagnosis, 12
Diastolic murmur of auricular fibrillation, 164
of mitral stenosis, 364
Diastolic pressure, 454
Diastolic wave in the jugular tracing, 469, 470
Dicrotic wave, 451
Digital examination of pulse, superiority of, 445
Digitalin granules, Nativelle's, 207
Digitalis, action of, 405
on heart, 69, 70
on vagus, 97, 246, 406
danger of, in auricular flutter, 246
diuretic action of, 206
dosage of, 207
effect of, in affections of genetic system, 293, 295
emetic action of, 97, 295
employment of, 208-211
in auricular fibrillation, 171, 173, 174, 179, 180, 204-213
in auricular flutter, 241-246
in myocardial affections, 312, 313
indications for use of, 205
method of administration of, 206
mode of action of, in auricular fibrillation, 205
in auricular flutter, 245
partial heart-block produced by, 245
sudden death from, 211-213

Dilatation of the aorta, 388, 389, *and see under* Aorta
of heart, 308-311
action of drugs in relation to, 310
and dropsy, 311, 312
and effort, 310, 311
and heart failure, 311
during strenuous bodily effort, 310, 311
from muscle failure, 311, 312
in acute affections of heart, 310
in mitral stenosis, 310
in relation to tone of heart, 308
in rheumatic fever, 394, 395
manner of production of, 308, 309
Diphtheria, heart in, 396
Disease, agents of, act as do therapeutic agents, 406
the recognition of, 3
Diseases, variety of, due to one morbid process, 404
Disorderly action of heart (D.A.H.), 9
Distress, cardiac, 410
Dropsy, 34
dilatation in relation to, 311, 312
in heart failure, 34
Drosera hairs, how influenced by chemical agents, 69
vital activities of, 52-54
Drugs and chemical agents, the action of, 69-71
Ductus arteriosus, patent, 380
murmur in, 381

Effector organ, the, 38, 39
Efficiency, pulse-rate as test of, 114
Effort and dilatation, 310, 311
effect of, in heart-block, 256, 257
on pulse-rate, 111, 112
limitation of, causes of, 410
with advancing age, 334, 335
Einthoven's string galvanometer, 424
Electrical stimulation as means of investigation, fallacy of, 48, 49, 103
Electricity, cell impulse and, 71, 72
Electro-cardiogram, the, 424-428
in auricular fibrillation, 168, 169, 192-194
in auricular flutter, 220
in paroxysmal tachycardia, 148
value of, in diagnosis, 12, 13, 108
Electro-cardiograph, the, 424
Electro-cardiographic waves due to auricle, 125
Embolism, cerebral, in mitral stenosis, 367, 368
Emetics, action of, 97
Empiricism of treatment, 405
End result, the, 36, 37
Endocarditis, infective, 396, 398, 399
diagnostic features of, 398
organisms in blood in, 398
symptoms and course of, 398, 399
malignant, 398
murmurs in, 392, 393
rheumatic, 394
septic, 396
symptoms in, 392, 393
Epigastric pulsation, 440-441
causes of, 440
significance of, 440
Eructation, gaseous, in angina pectoris, 338
Examination of the patient, 409-417
instrumental methods of, 418-428
methods of, 409-411
Excitability of genetic system, decreased, variations due to, 105
increased, as a cause of pain, 328
of nervous system, 328, 329
variations due to, 105
'Excitation wave', 426, 428
Exhaustion and the response to effort, 414, 415
clinical significance of sense of, 413
differentiation of, 412
due to ineffective heart-beat, 413, 414
due to vaso-motor disturbances, 414
in ill-health, 411, 412
mechanism of, 412
Experimental observations, value of, 405, 406
Extra-systole, the, 128
the auricular, 133
clinical diagnosis of the, 142
clinical methods of investigating, 128-130
definition of, 105
different forms of the, 133
electro-cardiogram of the, 133, 134
experimental methods of investigating, 130, 131
first recognition of, 6
the genetic system and the, 131-133
the interpolated, 140
the irregularity produced by the, 138
the nodal, 133
origin of the, 141, 142
pause in, 132, 140, 142
prognosis of the, 143-145
the ventricular, 133
Extra-systolic pulse-wave, size of the, 139, 140
Eye, capillary obliteration in the, 82, 83

Facies in aortic valvular disease, 375
in malignant endocarditis, 399
Fainting, 415-417
Fatty degeneration of heart muscle, 394
Febrile conditions, state of circulation in, 390, 391
Febrile illnesses, physiological murmurs in, 359
Fibrillation of muscle, 56, 75, 85
after nerve section, 60
Fibrillation waves, 168
Fibrotic change in tissues, in relation to capillary obliteration, 81
Fluctuation in relation to cutaneous sensation, 64-66
law of, 65
in relation to heart, 31
Foramen ovale, patent, 380
Functional efficiency of the heart, measuring the, 27-29

Functional impairment of organs ill understood, 27
Functional murmurs. *See under* Murmurs

Gallop rhythm, 357
Gaskell and his work on the heart, 8
Genetic and muscular systems, anatomy of, 90-92
  heart, relation between, 89
Genetic system of the heart, 39, 40, 84, 249, 250
  and the extra-systole, 131-133
  combined affections of the, 290-296
  decreased excitability of, 249-264
  effect of drugs on, 293-296
  evolution of, 60, 100
  function of the, 41, 60, 98-103
  in the production of death, 304, 305
  in relation to irregularities, 105-107
  increased excitability of, variations due to, 128-157
  independence of different parts, 125-127
  independent activity of, 106
  not a conducting system, 100
  relation of vagus and sympathetic nerves to, 96
  structure of, 57, 58, 100
  symptoms due to, 21
  vagus effect on, 296
Giddiness in heart-block, 262, 263
Graded action of heart muscle, 191, 192
Graphic and electro-cardiographic records, 108
Graphic records of auricular fibrillation, 166-169

Hæmoptysis from aneurysm, 386
  in mitral stenosis, 367
Hæmorrhage from aortic aneurysm, 384, 386, 387
Hairs of sundew, vital activities of, 52
Heart, acute infections of, dilatation in, 310
    management of, 400
    prognosis in, 399
    treatment in, 399, 400
  acute rheumatic affection of, symptoms of, 395, 396
Heart-beat, the, 84-89
  cessation of, 25
  parts concerned in the, 84, 85
Heart-block, 249-264
  adrenalin in, 264
  age-incidence of, 260
  associated with aortic valve disease, 259
  associated with arterial disease, 259
  associated with auricular fibrillation and flutter, 259
  associated with mitral stenosis, 258
  associated with myocardial disease, 259
  auricular fibrillation and, 249, 250, 259, 290-293
  cases illustrative of, 265-283
  clinical diagnosis of, 261
  complete, 255, 256
  conditions inducing, 256
Heart-block (*contd.*)—
  definition of, 251
  effect of atropine in, 257
  exhaustion in, 414
  in relation to the genetic system, 250
  in relation to heart failure, 261-263
  partial, 252, 253
    produced by digitalis, 245
  problem involved in, 250
  prognosis in, 263, 264
  response to effort in, 256, 257
  size of ventricular contraction in, 257
  treatment of, 264
Heart, blood supply of, in relation to its functional capacity, 25
  denervated, 96
  development of the, 85-89
  dilatation of, 308-311, *and see* Dilatation
  functional efficiency of, tests of, 27-29
  in acute affections, 390-400
  in infections, manner of affection of, 390, 391
  increased size of, in febrile affections, 391
    in myocardial affections, 306-308
  movements of the, 431-444
  position of, 429-431
Heart failure, and dilatation, 311
  and the response to effort, 30-35
  as test of prognostic value of heart signs, 6, 19, 24
  clinical records of, 32
  conditions inducing, in mitral regurgitation, 368, 369
  degrees of, 201
  early production of, 31
  enlarged liver in relation to, 477
  exhaustion in, 413, 414
  fluctuation in relation to, 65
  from chronic arsenical poisoning, 312
  from microbic infection, 312
  from myocardial affections, prognosis in, 312, 313
    treatment of, 313
  in auricular flutter, 238, 239
  in cases with history of rheumatic fever, 312
  in relation to arterial pressure, 458
  in relation to heart-block, 261-263
  manner of, with valvular defects, 362, 363
  mechanism of, 196, 197
  mitral stenosis in relation to, 363
  murmurs in relation to, 361
  origin of symptoms of, 31-33
  the production of, in auricular fibrillation, 196-198
  prognosis and, 24
  relation of, to small beats in auricular fibrillation, 194
  signs of, 30
  sudden recovery from, 197
  symptoms of, 197
    due to deficient output, 33-35
Heart muscle, condition of, in relation to prognosis, 200
  in relation to its functional capacity, 25

Heart-rate, changes in jugular tracing due to variation in, 470
in health, explanation of varying, 61
the normal, 110
Heart, rest and reserve power of, 30
Heart signs, prognostic value of, 9, 19
Heart sound, first, cause of, 356
first, modifications of, 356
second, origin of, 356
Heart sounds, abnormal, attitude of profession to, 355, 356
and murmurs, normal and abnormal, 354-361
change in, after onset of auricular fibrillation, 164
in auricular fibrillation, 163-166
variations in, 356, 357, *and see* Murmurs
'Heart strain', 162
Hemiplegia during febrile conditions, 393
Hydrothorax, 34
Hyperalgesia, 328
cutaneous, 328
experimental, 328
muscular, in enlarged liver, 478
Hyperpiesis, 457
Hypertrophy of heart, apex beat in, 434
in aortic stenosis, 372
in congenital heart affections, 380

Ill-health, the problem of, 36
Impulse, the cell, 49, 54
Impulses, delay in passage of, vital processes concerned in, 251
the discharge of, 67
the generation of, 98
in relation to cells, 38
natural, different from electrical, 49
originating in muscular system, 326
Infarcts from vegetations, 393
Infections, malignant, capillaries in, 26
Infective endocarditis. *See under* Endocarditis
Inhibition, as seen in hair of sundew leaf, 53
definition of, 49
Instrumental methods of examination, 418-428
Instrumental methods, the place of, 108, 109
Inter-systolic period, 251
Interval, the *a-c*, in tracings from neck, 252, *and see a-c* interval
presphygmic, 252
Inversion of electro-cardiographic waves, meaning of, 125
Investigation, principles of, 36, 37
Irregular pulse in auricular flutter, 218
Irregularities of heart, classification of, 104-107
early studies of, 6, 7, 20
in old age, 121
interpretation of, by aid of reflex arc, 42-46
Irregularity due to auricular fibrillation, 159, 160, 190, 191
due to extra-systole, 138
respiratory, presence of, as prognostic sign in acute febrile infections, 399
youthful type of, 20, 117, 118
clinical cases of, 20

Jaundice in enlarged liver, 481
Jugular pulse. *See* Pulse, jugular
Jugular tracing, method of analysing, 470-472

Kidney, functional efficiency of, tests of, 27
Kidneys, effects of capillary obliteration on, 81

Laboratory and clinical methods of diagnosis, 12, 19
Lactic acid, effect of, upon muscle of heart, 310
Laryngeal paralysis from aneurysm, 384, 385
Life insurance, importance of prognosis for, 15
Liver, enlargement of, 34
differential diagnosis of, 481, 482
reflex symptoms of, 477, 478
relation of, to heart failure, 477
signs of, 478
enlargement and pulsation of, 477-483
conditions producing, 479, 480
prognosis of, 482
treatment of, 483
movements due to cardiac aspiration, 439
pulsation of, 478, 479
different forms of, 481
in adhesive mediastino-pericarditis, 379
in tricuspid stenosis, 372
Localization of pain, 329
Lungs, œdema of, 34

Mammalian heart, impulse production and control in the, 88
Mediastino-pericarditis, adhesive, 377-379
ætiology of, 377
jugular pulse in, 378
prognosis of, 379
pulsus paradoxus in, 378
resection of ribs in, 379
symptoms of, 377-379
treatment of, 379
Medicine, modern, and prognosis, 14
need for new outlook in, 2, 22, 37
the science of, 1
the simplification of, 3
Medusa, controlled and uncontrolled movements in medusa, 62, 63
Mid-diastolic murmur, 364-366
Mitral pulse, the, 158
Mitral regurgitation, 368, 369
cause of, 368
in relation to heart failure, 368, 369
murmurs due to, 368
Mitral stenosis, 363-368
and heart-block, 258
cause of, 363
diastolic murmur of, 364
murmurs present in, 363-366
occasional symptoms of, 367, 368
progress and symptoms of, 366, 367
Morphine in anginal attacks, 345
Motor nerves, function of, 59
of bowel, 58
Movements of heart recognized by examination, 433

Murmur of aortic regurgitation, 373
of aortic stenosis, 372
auricular systolic, of Gairdner, 363
bellows, 373, 374
double aortic, 373, 374
mid-diastolic, 364-366
presystolic, 363, 364
quantity of blood thrown back to cause a, 358, 359
Murmurs, differentiation of functional and physiological from organic, 359, 360
functional, so-called, cause of, 357, 358
functional, so-called, significance of, 360, 361
in congenital heart affections, 380, 381
in infective endocarditis, 398
in mitral stenosis, 363-366
organic, significance of, 361
physiological, 357
significance of, 354, 355, 356
sounds and, normal and abnormal, 354-361
the timing of, 164
tricuspid, 370
Muscarin, effect of, on muscle of heart, 310
Muscle and nerve, relations of, 60
Muscle, contraction of, 55, 56
Muscle failure, 311, 312
Muscle fibre, description of, 56
Muscle of heart, characteristics of, 91
symptoms due to, 21
Muscles, impulses from, 326
pain from, 326, 327
Muscular and genetic systems of the heart, relation between, 89
Muscular contractions, effect of, on heart's rate, 94
Muscular effort, effect of, on auriculo-ventricular node, 189
Muscular fibres of heart, arrangement of, 92
Muscular system of the heart, 84, 85
Myocardial disease, and heart-block, 259
nature of symptoms of, 306
Myocarditis, rheumatic, 394
symptoms in, 391, 392
Myocardium, affections of, 306-313
treatment of, 313
in septic infections, 396

Nativelle's digitalin granules, 207
Nerve, relation of muscle to, 60
Nerve cells, structure and function of, 56, 57
Nerve supply of the heart, 93-97
Nerves, afferent system of, 93
and the capillaries, 78
functions of, 59
in relation to rate of recovery of muscle cells of heart, 58
Nervous system in relation to exhaustion, 413
Neurasthenia, 328
Neurone. *See* Nerve cell, 57
Neurones and muscle, 194
Nitrites in high blood-pressure, 460
Nitroglycerine in angina, 344, 345
'Nodal bradycardia', 291
Nodal rhythm. *See* Auricular fibrillation

Œdema of lungs, 34
Optic nerve and capillary supply, 82
Orthopnœa, 33

Pain, anginal, circumstances provoking, 335, 336
as a reaction, 67, 68
from muscle, 326, 327
imperfect knowledge of, 324
in aneurysm, 383, 388
in pericarditis, 393
localization of, 329
nature of, 325, 326
production of, rôle of sympathetic and vagus in, 94
structures concerned in production of, 330
value of, in diagnosis, 329, 330
in prognosis, 330, 331
Palpitation, 121, 122
Paroxysmal tachycardia. *See* Tachycardia
Pars membranacea, rôle of, 92
Pathology, a functional, needed, 401
principles of, 401-404
Pause, the, in extra-systole, 132, 140, 142
Pectinate fibres, function of, 92
Pericardial effusions, 394
Pericarditis, causes of, 393
effusion in, 393, 394
rheumatic, 394
symptoms in, 393, 394
Peripheral resistance, increased, effect of, on heart, 457, 458
Pharmacological research, 407, 408
Pneumonia, the heart in, 396
Polygraph, the, 419
clinical, the, 419, 421
the ink, 421-424
Presphygmic interval in cardiogram, 434
Presystolic murmur, disappearance of, 364
disappearance of, in auricular fibrillation, 164
Presystolic murmur of mitral stenosis, 363, 364
Principles of pathology and treatment, 401-408
Process, definition of, 49
Prognosis and heart failure, 24
and the multitude of symptoms, 23
in acute infections of the heart, 399
in angina pectoris, 340-342
in aortic aneurysm, 386, 387
in auricular fibrillation, 199-203
in auricular flutter, 239, 240
of the extra-systole, 143-145
in heart-block, 263, 264
of high blood-pressure, 458, 459
in liver enlargement and pulsation, 482
in myocardial affections, 312, 313
in the past, 14
of pulsus alternans, 288, 289
in relation to life insurance, 15
in relation to treatment, 13
in secondary angina, 349, 350

Prognosis (*contd.*)—
in sinus irregularities, 123, 124
in valvular affections, 376
the kind of, wanted, 24
modern medicine and, 14
the neglect of, 17, 18
new method for investigating, 21
of slow pulse, 116
object of, 13
the principles of, 17-26
reflex arc in relation to, 23
research into, 19
Prognostic symptoms in heart affections, sources of, 21
Propositions on which scientific classification of symptoms is based, 74
Puerperal septicæmia, heart infection in, 398
Pulmonary œdema in heart failure, 34
Pulsation, arterial, visible, 447
of aneurysm, 383, 384
of liver, 478, 479
Pulse, anacrotic, 372
character of the, 448
Corrigan's, 374
'Pulse deficit', 163
Pulse, digital examination of, superiority of, 445
effect of aortic aneurysm on, 385
examination of, 445-452
impact, 449
in acute infections of heart, 398
in acute pneumonia, 396
in auricular fibrillation, 163
in auricular flutter, 216-218
in myocarditis, 391, 392
jugular, in auricular fibrillation, 166, 167, 168
in heart-block, 261
inspection of, 461, 462
methods of recording, 462
recognition of events in, 462, 463
standards for interpreting, 466-468
liver, forms of, 479
method of examining, 447-449
mitral, 158
movement, explanation of, 445-447
nature of the, 445-447
rhythm of, 449
significance of, 461
the venous, 461-476
venous, anomalous forms of, 474
conditions giving rise to the, 474-476
significance of, 461
ventricular form of, 371, 472-474
ventricular form of, conditions producing, 473, 474
water-hammer, 374
Pulse-rate, 448
as a test of cardiac efficiency, 114
as measure of activity of sino-auricular node, 189
in fevers, 112
increased, 111-114
microbic infections in relation to, 113
significance of, 112, 113
Pulse-rate (*contd.*)—
normal, 110
slow, differentiation of forms of, 114, 115
factors concerned in, 114, 115
prognosis in, 116
recognition of forms of, by unaided senses, 115
with normal rhythm, 114
Pulse-wave, size of the, 448, 449
Pulses, radial, comparison of the, 449
Pulsus alternans, 284-289
cases illustrating, 222, 225, 289
clinical diagnosis of, 287
conditions favouring appearance of, 285, 286
definition of, 284
differential diagnosis of, 288
explanations of, 284, 285
in auricular flutter, 218
prognosis of, 288, 289
Pulsus bisferiens, 372
Pulsus celer, 449
Pulsus paradoxus in adhesive mediastino-pericarditis, 378
Pulsus tardus, 449
Purkinje cells or fibres, 58, 91
Purkinje tissue, structure of, 100, 101

Quinidine, danger of, 213
in auricular fibrillation, 213, 214

Radial beats, small, in paroxysmal tachycardia, 155
Radial pulse in auricular fibrillation, 167
Radial pulse-waves, variations in size of, 139, 140
Rate of heart, normal, 110
Receptors, 39
Reciprocal innervation, 61, 96
Reduplication of first sound, significance of, 356
of second sound, 356, 357
Reflex, definition of, a, 38
Reflex arc, the, 38-47
and prognosis, 23
and symptoms, 74
angina pectoris and the principle of the, 332, 333
application of the principle of the, 39, 41
as guide to investigation of symptoms, 75
constitution of, 38
for the interpretation of symptoms, 46
meaning of the, 38
Reflexes, cardiac, 94, 95
Regurgitation, mitral. *See* Mitral regurgitation
Respiration in relation to death, 25, 26
Respiratory variations in the youthful type of irregularity, 118
Response of heart to effort, 31
exhaustion and the, 414, 415
how its limitation is produced, 31
mechanism of increased heart rate in, 93, 94
methods of estimating, 410
Response to treatment in relation to prognosis, 200, 201

Retraction during ventricular systole as shown in cardiogram, 438, 439
Rheumatic fever, heart in, 394-396
Rhythm, idio-ventricular, 250
nodal. *See* Auricular fibrillation
the normal, 110-116
periodic variations in, 119, 120
of pulse, examination of, 449
Rhythmogenic function of the heart, 99, 100
Rhythms, abnormal, 106, 107
structures and processes concerned in, 107
Ribs, resection of, in adhesive mediastino-pericarditis, 379
Riva-Rocci sphygmomanometer, Martin modification of, 459
Rupture of heart, conditions causing, 309, 310

St. Andrews Institute for Clinical Research, objects of, 10, 37, 73
Salicylates in acute affections of the heart, 399
Salivation, in angina pectoris, 338
Sclerotic changes in heart from capillary obliteration, 80
Senile changes in the eye, 82, 83
Sensations, origin of, 67, 68
Senses, trained, importance of the, 18
unaided, training of the, 108, 109, 115, 116, 164-166
Septic infections, heart in, 396-399
Shock, capillaries in, 26
Sinking, sensation of, 412
Sino-auricular block, 257
Sino-auricular node, action of agents of disease upon, 112
disease of the, 182, 183
disturbances of the, 117-124
effects of loss of control of, 62
evidences of damage to, 183
exhaustion and recovery of, 41
functions of sympathetic and vagus nerves in relation to, 58, 61, 62
in relation to heart movements, 41, 42, 60, 61, 106
increased excitability of, not a sign of disease, 113
independent activity of, 106
its independence of auricles and ventricles, 96
liability of, to disturbance, 110
loss of influence of, 106, 161, 182
rôle of, 41, 75
structure of, 58, 90
structures controlled by, 75, 76
susceptibility of, to digitalis, 126
vagus and sympathetic control over, 62
vagus and sympathetic in relation to, 189, 190
variations in activity of cells of, in relation to pulse rate, 111
Sinus irregularities, 117-124
in old age, 121
prognosis in, 123, 124
Sinus venosus, 86
Soldier's heart, the, 351-353
early study of, 9, 10
Sphygmogram, as affected by instrumental defect, 451, 452
definition of, 450
diastolic period of, 451
systolic period of, 450
value of the, 190, 449, 450
Sphygmograph, the, 418, 419
Sphygmomanometer, forms of, 453-456
Spinal cord, effect of aneurysmal pressure on, 385, 386
Standardization of drugs, 207, 208
Standstill of the whole heart, 120, 121
'Stannius ligatures,' results of, 87, 88
Stimulation, definition of, 49
in hair of sundew leaf, 53
of structures as cause of pain, 327, 328
of tissues by mechanical means, 325
of tissues by vital processes, 325, 326
of the vagus or sympathetic, 97
Stimulus-conduction system of Tawara, 99
Stokes-Adams syndrome, 262, 264, 274
Stomach, functional efficiency of, tests of, 27
Sudden death, 201-203
from digitalis, 211
in angina pectoris, 338, 339, 340, 341
in diphtheria, 396
in pneumonia, 396
Sundew. *See* Drosera
Swallowing, accelerating effect of, on heart, 296
Sympathetic and vagus nerves, 95
relation of, to heart, 95, 96
rôle of, in relation to heart's rate, 94, 96
Sympathetic control over auriculo-ventricular node, 62
Sympathetic nerves, functions of, 58, 93
stimulation of the, 97
Symptoms, assessment of value of, 3, 23
basis for classification of, 49, 50
classification of, 73-76
common factor in, 402, 403
distinction between primary and secondary, 328, 329, 353
due to disorderly activity of organs, 181, 182
explanation of, 74
interpretation of, by reflex arc, 46
investigation of, guide to, 75
production of, 402
the significance of, 12-16, 23
three kinds of, 75
Syncopal attacks, 411
causes of, 415, 416
in auricular flutter, 238
Syncope, forms of, 415-416
Syphilis and the circulatory system, 319, 320
as cause of aneurysm, 388
Syphilitic heart affections, 319, 320
clinical records of, 320-323
Systolic plateau in cardiogram, 436
Systolic pressure, 454

Tachycardia, paroxysmal, clinical diagnosis of, 156
definition of, 105

Tachycardia (*contd.*)—
different forms of, 148-155
in mitral stenosis, 367
nature of attacks of, 147
origin of, 150, 151
prognosis of, 156
treatment of, 156
Tænia terminalis, function of, 92
Tapping for pericardial effusion, 394
Tests of functional efficiency of heart, 28
Therapeutics, backward state of, 69
Tone, 66, 85
definition of, 66, 102
in relation to viscero-motor reflex, 66
of heart muscle, 308, 310
of heart as a response to an impulse, 310
Toxins, action of, on heart, 390, 391, 394, 396
in relation to heart's rate, 113, 123
Tracheal tugging, 384
Treatment of acute infections of heart, 399, 400
of adhesive mediastino-pericarditis, 379
of aneurysm of aorta, 387, 388
of angina pectoris, 342-347
secondary, 350
of auricular fibrillation, 204-214
of auricular flutter, 241-248
of congenital heart affections, 381
of dilatation of aorta, 389
of extra-systole, 145, 146
of heart-block, 264
of high arterial pressure, 459, 460
of liver enlargement and pulsation, 483
of myocardial affections, 313
of paroxysmal tachycardia, 156
of valvular affections, 376
principles of, 404-408
Treatment, response to, as guide to prognosis, 200, 201, 240, 312
Tricuspid incompetence, 370, 371
Tricuspid murmurs, 370
Tricuspid regurgitation, 358
as shown in jugular tracing, 466
Tricuspid stenosis, 371, 372
auricular liver pulse in, 480
Tricuspid valves, affections of, 370-372
doubtful competency of, 370
opening of, as shown in jugular tracing, 465, 466
Trinitrin in angina, 344, 345
Tufnell diet in aneurysm, 388

Unconsciousness in heart-block, 262, 263
Urine, increased flow of, in angina pectoris, 338

Vagal activity in relation to heart-block, 257
Vagal effect upon genetic system, 296
Vagal syncope, 415, 416
Vagus, action of atropine on, 69
action of digitalis on, 69
control over auriculo-ventricular node, 62
nerves, functions of, 58
rôle of, in relation to heart, 95, 96
stimulation of the, 97
Valve disease in relation to prognosis, 200
Valve lesions *per se* not necessarily serious, 355
Valves, in malignant endocarditis, 81
liability of, to attack by infective organisms, 354
part played by, in producing ill-health, 354
symptoms due to, 21
Valvular affections, prognosis in, 376
Valvular defects, 362-375
cardiac muscle in relation to, 362
in relation to heart failure, 362
treatment of, 376
Variations of rate and rhythm, genetic system in relation to, 104-107
'Vaso-motor angina', 336
Vaso-motor disturbances in angina pectoris, 337, 338
Vaso-motor reactions as cause of pain, 327
Vegetations, 393
Veins, pulsation in, 461, 462
Venous pulse, the, 461. *See* Pulse, venous
Ventricle, contraction of the, 92
control of, by a.-v. node, 41
control of, by s.-a. node, 41
left, dilatation of, 310
slowed by digitalis, 245
Ventricular beat, influence of, on auricular, 256
Ventricular contraction in heart-block, size of, 257
origin of stimulus for, 249
rôle of auriculo-ventricular node in, 192
Ventricular fibrillation, 304, 305
as mode of death in angina, 339, 340, 342
Ventricular form of venous pulse, 472-474
conditions producing, 473, 474
Ventricular notch in the jugular tracing, 469
Ventricular systole, shock due to, 444
Ventricular wave and fall in the jugular tracing, 465, 466
Vertebræ, erosion of, from aneurysm, 385, 386
Vital activities, factors concerned in, 37
in the human body, 55-72
in plants, 51-54
method of investigating, 51
Vital process, definition of term, 49
Vital processes, the, 48-50, 74
concerned in pain, 325
Vomiting in angina pectoris, 338

War, investigation into soldier's heart during the, 352
Wave, dicrotic, in sphygmogram, 451
outflow remainder, in a sphygmogram, 451
papillary, in sphygmogram, 451
percussion, in sphygmogram, 451
predicrotic, in sphygmogram, 451
primary, in sphygmogram, 451
tidal, in sphygmogram, 451
Waves in sphygmogram, 450, 451

X-ray diagnosis of dilatation of aorta, 389
X-ray examination in diagnosis of aneurysm, 383, 384

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